

**Towards Intentional Relational Well-Being: Syndemic Contributions of Mental Health,
Trauma Exposure, and Sociodemographic Factors to Risk for Intimate Partner Violence
Victimization**

by

Sara F. Stein

A dissertation submitted in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy
(Social Work and Psychology)
in The University of Michigan
2021

Doctoral Committee:

Professor Sandra Graham-Bermann, Co-Chair
Professor Andrew Grogan-Kaylor, Co-Chair
Professor Todd I. Herrenkohl
Dr. Quyen Ngo, Hazelden Betty Ford Foundation
Clinical Professor Julie M. Ribaudo

Sara F. Stein

steinsf@umich.edu

ORCID ID: 0000-0003-3030-8949

© Sara F. Stein 2021

Dedication

To Carrie and Julie for seeing so clearly what I was unable to see and always holding steady with
your vast love and kindness.

Acknowledgments

Dissertation studies one and two were made possible through the grants from the Blue Cross Blue Shield of Michigan Foundation (Principal Investigator: Sandra Graham-Bermann). Study three was supported by the National Institute on Alcohol Abuse and Alcoholism (NIAAA; grant K23AA022641; PI: Quyen Ngo), the National Center for Advancing Translational Sciences of the National Institutes of Health (NIH; 2UL1TR000433), and the University of Michigan Injury Center, an Injury Control Research Center funded by the Centers for Disease Control and Prevention (CDC; grant 5R49CE002099). This dissertation research was also supported by the Institute for Social Research Robert Kahn Fellowship, John Longres Award in Psychology, Kellogg Fellowship in Children and Families, Edward S. Bordin Graduate Research Award, Institute of Research on Women and Gender Community of Scholars Fellowship, Irene and William Gambrill Fellowship, Bobbe and Jon Bridge Award, Albert C. Cain Research Award in Child Psychopathology, Institute of Research on Women and Gender Research Grant Scholarship and Creative Activities Focusing on Women and Gender, Joint Doctoral Program Grant, Blue Cross Blue Shield of Michigan Foundation's Student Program Grant, Center for Disease Control Injury Center Summer Fellowship, and the Carol Thiessen Mowbray Research Fund, received by Sara F. Stein.

This dissertation research was only possible thanks to the support of many different people. I am incredibly grateful for the support of my dissertation co-chairs **Drs. Andrew Grogan-Kaylor and Sandra Graham-Bermann**. Thank you for believing in and supporting me from the first day I interviewed with you back in 2013. **Andy**: thank you for meeting with me weekly and for your dedication and patience when talking through the numerous empirical and intellectual questions that arose throughout the duration of this work. I could always count on a few good laughs during our meetings and look forward to continuing to work together during the next phase of my career (and continuing to be impressed by your Stata and Zoom tricks). **Sandy**: thank you for teaching me the value of just “making things happen”. From data collection to running groups to publishing papers, your spirit and drive will forever be taken with me. I am also very thankful for the guidance and support of my committee members **Drs. Todd**

Herrenkohl and Quyen Ngo, and Julie Ribaud. **Todd:** thank you for your guidance and wisdom both on this dissertation research as well as on other projects. **Quyen:** They always says that Wellesley Sisters are for life; I just never imagined that one of them would come in the form of one of my committee members. Thank you for the numerous mentorship hours, intellectual debates/conversations, “figuring out” sessions, and writing meetings (especially the ones that included Ajishin). **Julie:** Words aren’t sufficient to capture my deep gratitude for all of your mentorship and support. From the very first time that I heard you speak as a guest lecturer in Sandy’s class I knew how special you and your work is. I’m delighted to have been able to collaborate in the numerous ways that we have and cannot wait to continue our work together. Thank you for seeing so deeply and for holding the space for me to join you in that, even when it feels like I may never get there.

I am incredibly grateful to the fellowship and support of many colleagues that worked on the research projects associated with this dissertation work. To **Drs. Maria Galano and Hannah Clark:** a special thanks for all of your heart and heard work to make the follow-up a reality and for the numerous hours writing, reading drafts, and working to get this work out to the world. To the **Child Violence and Trauma Lab research assistants:** without you none of this work would have been possible. A special thanks to **Molly Potel** who always helped to make the drives to interviews a little bit more amusing! I also want to acknowledge the families that made this research possible by participating in studies one and two described in this dissertation. To **Jessica Ramirez, MPH, MSW:** thank you for your friendship and support as we navigated so many challenging and fun times together! To **Linping Duan:** Thank you for all of your help managing the data and running analyses. To the **Ahimsa Lab research assistants:** thank you for your engaging conversations and deep examination of complex social issues. And a huge thanks to all of the Ahimsa Lab’s research participants.

I am very thankful for the wisdom and support of numerous clinical mentors and faculty members. To **Drs. Carrie Hatcher-Kay and Sharon Gold-Steinberg:** thank you for giving me the most exquisite trauma training that one could ever hope for. Without you two, I would have never gotten through the past few years and wouldn’t be a fraction of the clinician that I am now. Thank you for showing me a new way. To **Dr. Joe Himle:** Thank you for your cherished mentorship and guidance. I am so grateful for your willingness (and excitement) to be a sounding board for ideas and a place that I knew that I could always go to get some clarity on next steps. A

huge thanks to **Drs. Alison Miller and Maria Muzik** who have taken me under their wings over the past few years, shown me the numerous ways that academia can be full of life and healing potential, and have helped to make my postdoc dreams a reality. A huge thanks to my remaining clinical mentors: **Barb Eagle, LMSW, Marnie Leavitt LMSW, Dr. Kelley Callahan, Jenn Gardner, LICSW, Dr. Janina Fisher, and the Sensorimotor Psychotherapy Institute.** Finally, I am so grateful to **all of my clients** who have taught me so much over the past seven years.

I am also incredibly grateful for the love and support of my family. To **my parents:** thank you for always being there to read drafts and to push me to do my best. Thank you for helping me to know that even in the worst of times I can call and help will be available. To **Dr. Aaron Stein:** thank you for your love and support. There are somethings that only we can really understand and I'm so glad that you're the other half of the "we". To **Dr. Tori Holden:** I'm so grateful to get to say that you're my sister-in-law! I look forward to days of cooking and adventuring together! To **Sarah Cechvala:** For being the sister that I never had. Even though it was tragedy that brought us together, I am so thankful to get to be a part of your life now. To **Aunt Max:** For your generous heart and all of your loving support. And a loving thanks to all the remaining members of my extended family mentioned and to **Colleen and Tony Corte.**

I am also thankful for all the love and support of my friends. To **my clinical cohort:** Drs. Elizabeth Yu, Andrew Pomerville, Cecilia Votta, and Ka IP – I can't believe that we all made it! **Dr. Elizabeth Yu:** thank you for your steady presence and friendship. From Ajishin to Disney Land to discount dogs to babies, I'm so happy to be able to call you a friend. To **Jenny Cleary:** Thank you for all of the serious talks, helping me to learn that silence is okay, the walks with doggos, the dinner making decisions, and the necessary (but excruciating) learning to giraffe together! To **Dr. Paige Safyer:** Thank you for spearheading the JR fan club (Madame President), for the endless hours debating attachment, for making Cecilia (and now Georgie) come to life, for showing me all of the ropes of the Joint Program, and for believing (and helping me to make a reality) some of my crazy ideas. So much more is yet to come. To **Dr. Lauren Bader:** For all of your loving spirit and always making me laugh. There is no one else that I'd rather share both a stats class and a yoga class with (or be locked out of an Airbnb in Rome with). To **Virginia Lodge:** Dear, there aren't words for how much you mean to me! Thank you for being you! I can't wait to continue to know you for a very long time. **Kristin Kunes and Sarah Meier:**

Thank you for your deep love, wisdom, and friendship. The Fantastic Four will live on forever! To **Rachel Burrage**: For taking me under your wing even before I started the Joint Program. Thank you for your mentorship and friendship. To my **Blossom Sangha**: Thank you for your love and for the opportunity to just being real together. Finally to **Lauren Kenny, Aaron Siegfried, Anne Blumenthal, Charlene Mangi, Ita Reyes, Erin Kelley, Katie Kelly, Nancy Boyd, and many others not named here.**

And finally, I am so grateful for my little furry ones. To **Luis Miguel and Ainsworth**: you mean the absolute world to me. Thank you for being a source of such incredible light and love. May we walk forever in joyous company together. <3

Table of Contents

Dedication.....	ii
Acknowledgments.....	iii
List of Tables.....	ix
List of Figures	x
List of Appendices	xi
Abstract	xii
Chapter 1	1
Introduction	1
Theoretical Framework.....	3
Syndemic Factors of Mental Health and Their Relationship to IPV	6
Syndemic Factors of Trauma Exposure and Relationship to IPV	13
Sociodemographic Factors and Relationship to IPV	19
Major Conclusions and Limitations of Extant Research	22
The Dissertation Studies	24
Chapter II.....	26
The Contributions of the Posttraumatic Stress Symptom Domains to Intimate Partner Violence Victimization Across Eight Years	26
Method	32
Results	39
Discussion.....	41
Chapter III	53
Predictors of Intimate Partner Violence Victimization by Multiple Partners over Eight Years ..	53
Method	59
Results	67
Discussion.....	69
Chapter IV	84
Predictors of Experiencing Intimate Partner Violence Victimization in a Community Sample of Emerging Adults.....	84
Methods	91
Results	96
Discussion.....	98

Chapter V	117
Conclusion.....	117
The Dissertation Studies	118
Clinical Implications.....	123
Limitations	124
Future directions	127
Summary.....	132
References	134
Appendices	168

List of Tables

Table 2.1. Descriptive statistics across time.	48
Table 2.2. Predictors of risk for IPV victimization across eight years.	49
Table 3.1. Study variables over the three measurement occasions.	77
Table 3.2. Post hoc analysis: Linear MLM model estimating re-engagement over eight years using indicator subscales.	78
Table 3.3. Correlation matrix of primary study variables (Supplemental table).	80
Table 3.4. Correlation matrix of study variables used in post-hoc analysis (supplemental table).	81
Table 3.5. Linear multilevel model estimating re-engagement over eight years (supplemental table).	83
Table 4.1. Sample descriptive statistics by victimization group (N=645).	108
Table 4.2. Model 1a: Logistic regression examining syndemic predictors of IPV victimization: NPV victimization * anxiety.	109
Table 4.3. Model 1b: Logistic regression examining syndemic predictors of IPV victimization: NPV victimization * IPV aggression interaction.	110
Table 4.4. Model 1c: Logistic regression examining syndemic predictors of IPV victimization: NPV victimization * alcohol use interaction.	111
Table 4.5. Model 1d: Logistic regression examining syndemic predictors of IPV victimization: NPV victimization * marijuana use.	112
Table 4.6. Model 2: Logistic regression predicting IPV victimization with an alcohol use * IPV aggression interaction.	113
Table 4.7. Model 3: Logistic regression predicting IPV victimization with an alcohol use * marijuana use interaction.	115

List of Figures

Figure 1.1. General concept model.	6
Figure 1.2. Conceptual flow of the three dissertation studies.	24
Figure 1.3. Dissertation conceptual model.	25
Figure 2.1. Interaction between time (weeks) and total PTS symptoms.	50
Figure 2.2. Interaction between time (weeks) and PTS arousal.	51
Figure 2.3. Interaction between time (weeks) and PTS reexperiencing.	52
Figure 3.1. Moms' Empowerment Program CONSORT flow diagram.	79
Figure 4.1. Interaction between IPV aggression and alcohol use on IPV victimization probability.	114
Figure 4.2. Interaction between alcohol and marijuana use on IPV victimization probability.	116

List of Appendices

Appendix A. Demographic questionnaire (Study 1 & 2).	168
Appendix B. Revised Conflict Tactics Scale (Study 1 & 2).	170
Appendix C. Posttraumatic Diagnostic Scale (Study 1 & 2).	172
Appendix D. Center for Epidemiologic Studies Depression Scale (Study 1 & 2).	175

Abstract

Treatments for reducing intimate partner violence (IPV) victimization have shown limited effectiveness, raising questions about the appropriateness of the mechanisms of risk targeted through these interventions. Yet, limited empirical work has delineated said mechanism of risk. A comprehensive understanding of these mechanisms is required to effectively mitigate future victimization. The aim of this dissertation is to contribute empirical evidence regarding modifiable factors that a person can address to change their intimate relationship trajectories and work towards intentional relational well-being. This research utilizes a syndemics framework to identify the trauma exposure, mental health, and sociodemographic contributors to IPV victimization risk across three independent studies.

Study one follows women across eight years to identify the risk factors for the total amount of IPV victimization experienced. Higher levels of posttraumatic stress (PTS) symptoms were associated with initially greater IPV victimization. However, across time women with higher PTS symptoms decreased more quickly in the amount of IPV victimization than those with lower PTS symptoms. Higher levels of PTS arousal and reexperiencing were associated with initially higher levels of IPV victimization. However, over time women with high levels of PTS arousal decreased more quickly in the amount of IPV victimization than those with low levels of PTS arousal. This was not the case for reexperiencing: higher levels of PTS reexperiencing started off and remained associated with higher levels of IPV victimization across time. Age was inversely related to IPV victimization only when accounting for the PTS

symptom domains. Findings suggest that collapsing PTS symptoms into an overall construct may be too imprecise to identify key mechanisms of IPV victimization risk. Intervention efforts should prioritize addressing reexperiencing symptoms as a pathway to limiting future IPV victimization.

Study two follows women over eight years to delineate the risk factors for IPV re-engagement (number of violent partners). More psychological but less sexual IPV was associated with greater re-engagement. A greater number of PTS reexperiencing symptoms were associated with less re-engagement. Lower levels of positive affect and higher somatic symptoms (depressive symptoms) were associated with higher re-engagement. Higher income and lower housing instability were associated with more re-engagement. Findings suggest that it is not what happened that creates risk for re-engagement, but rather how women are doing following traumatic experiences.

Study three examines the role trauma exposure and mental health on the odds of experiencing IPV victimization in emerging adults. Non-partner violence victimization was not associated with IPV victimization, nor were anxiety or marijuana use alone. However, IPV aggression, alcohol consumption, and identification as female were associated with IPV victimization. Findings suggested a synergistic relationship between IPV aggression and alcohol use and alcohol and marijuana use on IPV victimization. At low levels of IPV aggression (1-3 acts in the prior six months), alcohol use was associated with increased IPV victimization risk, but this did not hold for four acts or more. Here, IPV victimization risk remained high across all levels of alcohol consumption. Furthermore, those who did not use marijuana had significantly higher IPV victimization risk as alcohol use increased. This was not true of those who did use marijuana as IPV victimization risk did not increase as alcohol use increased. Findings suggest

that alcohol use is an important but insufficient predictor of IPV victimization alone and needs to be considered in tandem with other indicators of risk.

Chapter 1

Introduction

Intimate partner violence (IPV) is a pervasive social issue that includes the acts or threats of physical, sexual, and emotional violence by a current or former intimate partner, encompassing stalking, psychological aggression, and coercive behavior tactics (Black et al., 2011). Findings from the Center for Disease Control's 2015 National Intimate Partner and Sexual Violence Survey (S. G. Smith et al., 2018) suggest that approximately 36% of women and 34% of men will experience IPV in their lifetime (including physical violence, sexual violence, and/or stalking). Women also experience high rates of physical IPV with nearly 32% experiencing some form of physical IPV in their lifetime; additionally, 22% of women are estimated to experience at least one act of severe physical violence by an intimate partner across their lifespan (Breiding et al., 2014). Furthermore, 18% of women experience sexual violence by an intimate partner (S. G. Smith et al., 2018). Rates of psychological violence are the highest with experiences of psychological aggression by an intimate partner occurring for 47% of women across their lifetime (Breiding et al., 2014).

Existing literature further demonstrates that IPV victimization in women often occurs across more than a single intimate partner (Alexander, 2009; Coolidge & Anderson, 2002; S. F. Stein et al., 2019). Documented re-engagement rates for women who have experienced IPV victimization range from 35 - 56% (Alexander, 2009; Cole, Logan, & Shannon, 2008; Stein et al., 2016). In addition to the high prevalence of IPV victimization in women and across partnerships, recent findings suggest that bidirectional or mutual IPV may account for the

majority of violence between intimate partners, with a comprehensive review of the literature revealing that 57.9% of IPV is bidirectional (Langhinrichsen-Rohling et al., 2012). However, women have been found to have poorer outcomes as compared to men as a result of IPV victimization partially due to contextual factors relating to power and control that may exacerbate the effects of the traumatic exposure (Caldwell et al., 2012).

The economic costs of IPV are high with recent estimates using the U.S. National Intimate Partner Violence and Sexual Violence Survey data showing that the lifetime economic cost of experiencing IPV is more than \$100,000 per woman and \$23,000 per man (Peterson et al., 2018). This amounts to a population lifetime economic burden of \$3.6 trillion, 59% of which is direct medical costs. Women who have experienced IPV have higher lifetime rates of health care utilization than women without IPV victimization, including hospital visits (10.2% of women with IPV victimization compared to 8.3% of women without IPV victimization), emergency department visits (5.9% of women with IPV victimization; 3.8% of women without), inpatient admissions (5.3% of women with IPV victimization; 4.7% of women without), and mental health services (13.7% women with IPV victimization; 7.3% for women without; Rivara et al., 2007). Lifetime lost wages from employment interruptions associated with IPV involvement further account for \$1.3 trillion and an additional \$73 billion is spent on criminal justice related activities (Peterson et al., 2018). The social burden of these costs is significant as government sources paid for more than one-third of the \$3.6 trillion IPV population cost.

In addition to the economic costs, IPV victimization has known severe negative physical and mental health consequences including post-traumatic stress (PTS), depression, anxiety, substance use problems, and overall poor physical health (Lacey et al., 2012; Lagdon et al., 2014; Loxton et al., 2017; Ouellet-Morin et al., 2015). Posttraumatic stress is one of the most

frequent outcomes of experiencing IPV (Dutton et al., 2006; Ehrensaft, Moffitt, & Caspi, 2006; Golding, 1999). In a seminal meta-analysis, Golding (1999) found that almost 64% of women who had experienced IPV victimization met criteria for a PTSD diagnosis. Depression is also a significant sequela of IPV victimization: in a systematic review and meta-analysis, women who had experienced IPV were found to have had a two to three-fold increased risk of major depressive disorder and a 1.5 to two-fold increase in depressive symptoms compared to women without IPV victimization (Beydoun et al., 2012). Prior research has also suggested a dosing relationship for IPV with PTS and depression such that greater amounts of IPV are associated with severity of symptoms (Golding, 1999). IPV victimization has also been linked to increased anxiety, with a recent study finding a nearly 2-fold increase in odds of having anxiety for women with IPV victimization compared to those without (Chandan et al., 2019). Substance use is a further documented sequela of IPV victimization: Ouellet-Morin and colleagues (2015), using a sample of 1,052 mothers from a nationally representative study, showed that women who have experienced IPV victimization have high rates of alcohol use. Dissatisfaction in relationships and relationship instability are additional long-term outcomes of IPV victimization in women (Simmons et al., 2018).

As evidenced here, the social and individual costs of IPV victimization are enormous. It is thus a social imperative to develop evidenced-based, targeted strategies and interventions to reduce and mitigate the endemic occurrence of violence between intimate partners. Although research has long aimed to identify and understand different components of risk for IPV perpetration, especially in men, treatment programs remain largely ineffective at reducing recidivism (Babcock et al., 2004; Eckhardt et al., 2013). Therefore, another important avenue for

exploration to address this crucial social problem is the identification of the contributors of vulnerability for experiencing IPV victimization.

Theoretical Framework

Evidence suggests that IPV is a dyadic and relational process, meaning that characteristics of both the person perpetrating and the person experiencing the IPV victimization may contribute risk to the occurrence of violence between intimate partners (Kuijpers, Van der Knaap, et al., 2012b; Moffitt et al., 2001). Theoretical frameworks such as the Developmental Systems Perspective on aggression towards a partner (Capaldi et al., 2005; Capaldi & Kim, 2007) also indicate that there may be factors in women's lives that may increase their vulnerability to IPV victimization. Although explicating the issue of risk factors for IPV victimization can be a precarious conversation and has sometimes been criticized as "victim blaming" (Cattaneo & Goodman, 2005), ignoring these factors is a limited and dangerous perspective as it supports the notion of those who have experienced IPV victimization as helpless objects with no agency in their interpersonal interactions and relationships. Furthermore, it fails to identify potentially modifiable risk factors that may be important components of intervention. Thus, empirical work has documented the importance of identifying factors that those experiencing IPV victimization can influence to reduce future risk for victimization (Goodman et al., 2005; Kuijpers et al., 2011; Perez & Johnson, 2008). A clear understanding of the contributing mechanisms of vulnerability for IPV victimization will allow for the development of prevention programs and treatment interventions that specifically target these risk factors as a pathway to effective and sustained IPV victimization reduction and mitigation. Interventions that target IPV victimization risk could help individuals foster a greater sense of personal power and autonomy in selecting and establishing healthy and rewarding intimate interpersonal

relationships. The overarching aim of this work is thus to contribute empirical evidence for modifiable individual and social factors that a person can address in the journey towards intentional relational wellbeing.

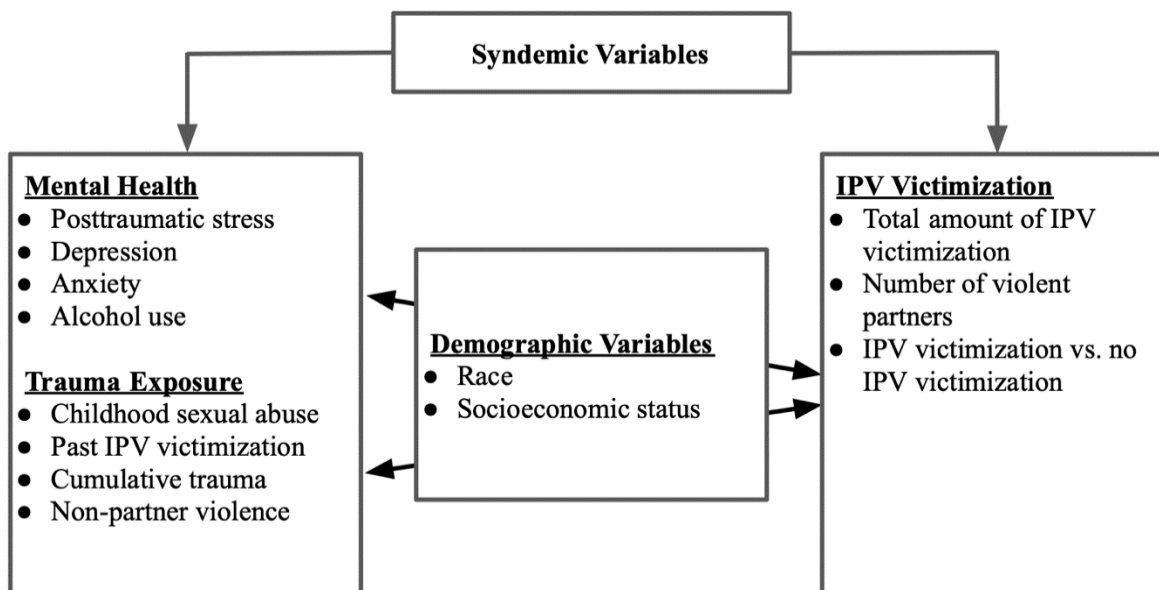
For decades, researchers have worked to understand and explain how and why IPV occurs, an effort that has resulted in several competing explanatory frameworks such as second-wave Feminist Theory (Dobash & Dobash, 1979), Family Violence Perspective (Gelles & Straus, 1979; Giles-Sims, 1983; Straus, 1973), Social Learning Theory (Bandura, 1978), and the Typology of IPV (Johnson, 1995, 2008), amongst others. However, despite the enormous theoretical and empirical contributions of these and other frameworks to explain aspects of the occurrence of IPV, individually they are limited in scope and unable to provide a complex and comprehensive understanding of the many individual and social factors that contribute to IPV victimization risk (Bell & Naugle, 2014). The theory of syndemics or synergistic epidemics (Singer, 2009) integrates elements of the above-mentioned theories and offers a more comprehensive framework for beginning to delineate the complexity of IPV victimization risk.

Although originally developed as a framework for understanding sequential epidemics or disease clusters (Singer, 2009), syndemics has been widely applied to social problems and socio-behavioral outcomes (Klein, 2011), such as IPV, mental health concerns, and trauma (Brezing et al., 2015; Illangasekare et al., 2013; Meyer et al., 2011; Senn et al., 2010; Sullivan et al., 2015). Syndemics purports that diseases or epidemics overlap and mutually contribute to one another, resulting in both the clustering of and interaction between said epidemics (Singer, 2009; Singer, Bulled, Ostrach, & Mendenhall, 2017; Singer & Clair, 2003). Central to the theory is the idea that the social contexts in which these interactions occur fundamentally contribute to and exacerbate how the epidemics interact. Human social contexts or environments are comprised of

dominant structures of social relationships, such as social inequality and injustice, which profoundly contribute to and aggravate the clustering and overlap of social problems (Singer, 2009; Singer, Bulled, Ostrach, & Mendenhall, 2017). Thus, syndemics provides a concurrent “disease” to “disease” and social context to “disease” interaction perspective (Singer et al., 2017), which is a comprehensive framework for examining and explicating IPV victimization risk.

Decades of empirical work has evidenced a consistent overlap in three epidemics: IPV victimization, mental health concerns (e.g. traumatic stress, depression, anxiety, and alcohol use; Ehrensaft et al., 2006), and trauma exposure (e.g. childhood sexual abuse, prior IPV, cumulative trauma exposure, and non-partner violence; Daigneault, Hébert, & McDuff, 2009; Whitfield, Anda, Dube, & Felitti, 2003), suggesting a synergistic or syndemic relationship between these three social problems. Furthermore, social contextual factors (e.g. race and socioeconomic status) have been linked to differing rates of all three epidemics and severity outcomes (Golden et al., 2013; James et al., 2013; Lipsky et al., 2009). This dissertation seeks to explore and identify potential contributors to IPV victimization risk using a syndemics framework, concurrently considering the epidemics of mental health concerns and trauma exposure and sociodemographic factors as potential contributors to IPV victimization vulnerability as represented in the General Concept Model below (See Figure 1.1).

Figure 1.1. General Concept Model



The following sections will explore the existing empirical evidence for the synergistic relationship between mental health concerns, trauma exposure, and sociodemographic factors and IPV victimization.

Syndemic Factors of Mental Health and Their Relationship to IPV

Posttraumatic Stress

Mental health concerns represent a known risk factor for involvement in IPV (Ehrensaft et al., 2006). Posttraumatic stress disorder (PTSD) is a concern associated with the dysregulation of the nervous system that may occur following exposure to a traumatic event or events (A. N. Schore, 2002). Symptom domains of posttraumatic stress (PTS) required to meet diagnostic criteria for PTSD in the DSM-5 (American Psychiatric Association, 2013) include: reexperiencing (e.g. somatic or image-based flashbacks), avoidance (e.g. avoidance of distressing memories, thoughts, feelings, or external reminders of the traumatic event), negative alternations in cognitions and mood (e.g. feeling isolated), and arousal (e.g. hypervigilance). Individuals are said to have PTS when they exhibit symptoms of PTS but do not necessarily meet

full criteria for a diagnosis of PTSD. Posttraumatic stress symptoms are not only a known outcome of experiencing IPV victimization but have also been linked to risk for IPV victimization (Iverson et al., 2011; Krause, Kaltman, Goodman, & Dutton, 2006; Perez & Johnson, 2008). Posttraumatic stress may compromise an individual's ability to accurately read, assess, and respond to environmental danger cues using present-moment data (Iverson et al., 2011; Van der Kolk, 1994, 2015). In the context of IPV, PTS may impede the capacity to correctly and effectively attend and respond to signs of threat from an intimate partner (Iverson et al., 2011).

Overall, extant research has found a somewhat inconsistent link between PTS and future victimization to IPV (Krause et al., 2006; Perez & Johnson, 2008; Sonis & Langer, 2008; S. F. Stein et al., 2019). Some longitudinal evidence has clearly shown that PTS is associated with greater IPV victimization. For example, women who had continued IPV victimization over one year had significantly more PTSD symptoms than those who did not experience IPV during that time (Krause et al., 2006). IPV victimization was operationalized to include physical IPV, sexual coercion by an intimate partner, and IPV-related injury. In another study, reductions in PTSD symptoms were associated with reduced likelihood of physical IPV victimization at a six-month follow-up in a sample of 150 women with a PTSD diagnosis at baseline (Iverson et al., 2011). In contrast, other research has not identified a strong link between PTS and IPV victimization (Cole et al., 2008; Sonis, 2008). PTS was not associated with women's engagement with multiple violent intimate partners, in a sample of 164 women who had experienced IPV victimization, (S. F. Stein et al., 2019). A limitation of this work, however, is that it used cross-sectional data and therefore the temporal dynamics of the conclusion remain unknown. In other work examining the accuracy with which women were able to assess future IPV victimization using a sample of

women with PTSD (Bell, Cattaneo, Goodman, & Dutton, 2008), a single standard deviation increase in the number of PTS symptoms was associated with 1.68 increase in the accuracy odds of women predicting future IPV victimization compared to those with no IPV 18 months later, suggesting that there are ways that PTS may be protective against future victimization. The inconsistencies in the findings may be partially accounted for by discrepant conceptualizations of IPV victimization in each study. Continued investigation using longitudinal data over a longer period of time and more comprehensive operationalizations of IPV victimization (including physical, sexual, and psychological IPV and IPV re-engagement) are needed to more accurately understand the contributions of PTS to IPV victimization risk.

The inconsistencies in the findings may also indicate that there may be aspects of PTS that may be protective against future IPV victimization. The presentation of PTSD is nuanced and includes seemingly disparate types of symptomatology (Van der Kolk, 1994). For example, numbing and hyperarousal are both symptoms frequently seen in the presentation of PTS but are physiologically and experientially very different. Numbing includes a potentially blunted responsiveness to the environment while hyperarousal may increase sensitivity to trauma-related stimuli. It therefore follows that different PTS symptom domains may differentially contribute risk (or protection) to future IPV victimization. However, very limited work has examined the contributions of the specific domains on IPV victimization (Dutton, 2009; Iverson et al., 2013; Krause, Kaltman, Goodman, & Dutton, 2006). In a 6-month longitudinal study, hyperarousal symptoms but not numbing, avoidance, or reexperiencing predicted experiencing further physical IPV in women (Iverson et al., 2013). In contrast, another study found that PTS reexperiencing was associated with increased physical and psychological IPV over a 6-month time period (Kuijpers, van der Knaap, et al., 2012). Yet another investigation identified that PTS numbing

increased women's odds of experiencing further IPV by 1.49 times over a year period, which equates to a three-fold increase in IPV victimization odds when shifting from endorsing no numbing symptoms to two (Krause et al., 2006). In further work, avoidance/numbing, reexperiencing, nor arousal predicted future IPV victimization (Cougle et al., 2009). Despite a prior call for continued research to understand the contributions of the PTS symptom domains to IPV victimization risk (Dutton, 2009), the empirical evidence remains very limited and inconclusive as shown here. Further research using longitudinal data over a longer period of time and more comprehensive operationalizations of IPV victimization (including physical, sexual, and psychological IPV and IPV re-engagement) are needed to delineate the contributions of each PTS symptom domain to IPV victimization risk.

Depression

Depression is another mental health concern that has been examined as a potential risk factor for future IPV victimization. Prior work has hypothesized that depressive symptoms in women may compromise the evaluation of threat in the environment and interpersonal relationships due to reduced cognitive and emotional capacity and may affect the ability to leave a violent situation or a violent partner due to high levels of guilt and hopelessness and low energy and motivation (Cougle et al., 2009; Iverson et al., 2011). However, the findings are mixed regarding the nature of the relationship between depression and IPV victimization (Iverson et al., 2011; Kuijpers et al., 2011).

Some empirical work has established a link between depression and future IPV victimization (Caetano, Vaeth, & Ramisetty-Mikler, 2008; Cougle et al., 2009; Iverson et al., 2011; Lehrer, Buka, Gortmaker, & Shrier, 2006). In a national probability sample of 2,863 women, major depression was found to increase the odds of new IPV victimization by 1.69 times

over two years (Cougler et al., 2009). Iverson and colleagues (2011) found that reductions in depression were significantly associated with lower levels of physical IPV victimization over 6-months. Women's depressive symptoms have also been linked to their involvement in multiple violent relationships, as women with more than one violent partner have been found to be nearly three times as likely as women with a single partner to meet diagnostic criteria for depression (Coolidge & Anderson, 2002). Still, other studies have failed to find that depression is predictive of IPV victimization (Cole et al., 2008; Perez & Johnson, 2008; Renner & Whitney, 2012; Sonis & Langer, 2008; Stein et al., 2016). For example, depression in the two weeks prior to baseline measurement was not associated with experiencing IPV with a new partner over the subsequent year in a study of 756 women who had obtained a domestic violence order (DVO) against an intimate partner (Cole et al., 2008). Furthermore, Stein and colleagues (2016) failed to find a relationship between depressive symptoms and women's engagement with multiple violent intimate partners. Continued research using prospective longitudinal evidence is needed to discern the potential role that depression may play in creating risk for IPV victimization given the very mixed nature of the extant findings.

Anxiety

Anxiety is an understudied potential mental health outcome of experiencing IPV (Golding, 1999; Kuijpers et al., 2011). Limited work has documented a link between IPV victimization and anxiety in women. For example, using the Fragile Family and Child Wellbeing data, Suglia, Duarte, and Sandel (2011) found that having experienced IPV victimization in the prior 12 months was associated with 2.92 times the odds of having generalized anxiety disorder compared to those who had not. A systematic review and meta-analysis (Trevillion et al., 2012) found that the pooled odds ratio for lifetime IPV victimization was 4.08 for among women with

anxiety disorders as compared to those without. Further, in a more recent population-based study in the UK (Chandan et al., 2019), women who had experienced IPV victimization had an 1.99 adjusted incidence rate ratio of anxiety increase compared to those without. Unfortunately, to date, no work has examined, the potential predictive role of anxiety in IPV victimization risk. In a systematic review of the prospective evidence for IPV revictimization, Kuijpers and colleagues (2011) were unable to identify any studies that examined the potential role of anxiety in IPV victimization risk. Empirical research on anxiety as a potential risk factor for IPV victimization is needed.

Alcohol Use

The issue of alcohol's role as a predictor of IPV has been controversial; feminists and women's advocacy groups have been reticent to consider the role of substances in women's victimization out of fear that alcohol will be seen as an excuse for IPV (Foran & Leary, 2008). Even so, a long history of research supports the idea that alcohol causes general aggression (Bushman & Cooper, 1990; Chermack & Giancola, 1997). Perpetrators' and victims' alcohol use has been widely examined as a risk factor in the literature for IPV victimization in women (Chermack & Giancola, 1997; Lipsey, Wilson, Cohen, & Derzon, 1997). Findings have highlighted a strong connection between alcohol use and IPV perpetration, with the focus historically having been on men (Chermack & Giancola, 1997; Hofeller, 1982; Glenda Kaufman Kantor & Straus, 1987; Lipsey et al., 1997; Roy, 1982). In a five-year longitudinal examination of couples in the United States, Caetano, McGrath, Ramisetty-Mikler, and Field (2005) identified that the volume of men's drinking was significantly associated with male to female partner violence (MFPV). In addition, male-drinking problems predicted higher overall MFPV and greater reoccurrence of MFPV. A more recent study found that alcohol abuse/dependence

increased the odds of IPV perpetration (in both men and women) by almost 2 times (Afifi et al., 2012).

The evidence surrounding women's alcohol use as a predictor of their victimization is much more mixed (Anderson, 2002; Kuijpers et al., 2011; Testa, 2004; White & Chen, 2002). Extant research has identified high levels of IPV among substance use clinical samples of women (Chermack et al., 2000) and high levels of alcohol use among individuals residing in domestic violence shelters (Kaysen et al., 2008; Martin et al., 2008). For example, in a systematic review, an estimated 22-72% of women in shelters were found to have a current or past problem with alcohol (Schumacher & Holt, 2012), however these studies on shelters are limited by a lack of standardized measures to assess alcohol use. Furthermore, results of a nationally representative sample of 1,052 showed that mothers who have experienced IPV have higher rates of alcohol use than those who have not (Ouellet-Morin et al., 2015).

A large body of evidence suggests some relationship between women's alcohol use (and abuse) and some type of IPV victimization, including sexual, physical, and psychological violence (Baker & Stith, 2008; Bowling, 2007; Cole et al., 2008; Golinelli, Longshore, & Wenzel, 2009; La Flair et al., 2012; Nowotny & Graves, 2013; Ouellet-Morin et al., 2015; Shorey, Rhatigan, Fite, & Stuart, 2011; Stuart, Moore, Ramsey, & Kahler, 2004; Temple, Weston, Stuart, & Marshall, 2008). For example, Vatnar and Bjørkly (2008) found that women's substance use was related to physical and psychological victimization, but not to sexual victimization. Furthermore, Fowler (2007) showed that women's number of years of alcohol use was significantly related to experiencing IPV physical abuse victimization. In contrast, Nowotny and Graves (2013) found that substance use was only associated with IPV victimization when the sample was analyzed by racial groups. Specifically, support was found for alcohol as a predictor

of IPV victimization in Black and Latina women, but not for White counterparts. Important limitations of these studies include inconsistent use of standardized measures to assess alcohol use and IPV victimization and retrospective designs (for an exception, see Temple and colleagues (2008)).

Other studies have not found support for any association between substance use and IPV victimization though the directionality of the relationship remains unclear (Gilbert, El-Bassel, Chang, Wu, & Roy, 2012; Kantor & Asdigian, 1997; La Flair et al., 2012; Lipsky, Caetano, Field, & Bazargan, 2005; Martino, Collins, & Ellickson, 2005; Poole, Greaves, Jategaonkar, McCullough, & Chabot, 2008; Testa, Livingston, & Leonard, 2004). In a longitudinal study of a representative sample of low-income women in an emergency department, binge drinking at baseline did not predict any type of subsequent IPV victimization at 6- or 12-month follow-up (Gilbert et al., 2012). Likewise, Renner and Whitney (2012) found no significant relationship between alcohol use and IPV victimization using a sample of young adults from the National Longitudinal Study of Adolescent Health.

The empirical evidence on women's alcohol use and their IPV victimization risk remains too mixed to be able to reach any substantial conclusion. It is possible that variation in measurement of alcohol use and IPV may, in part, account for these varied findings. Continued work using consistent and standardized measures for alcohol and IPV is required to discern the role that alcohol may play in contributing risk to IPV victimization.

The next sections review the literature on exposure to prior traumatic events as risk factors for IPV victimization. These include childhood sexual abuse, prior IPV victimization, and cumulative trauma.

Syndemic Factors of Trauma Exposure and Relationship to IPV

Childhood sexual abuse

Scholars have long been interested in the role of childhood abuse in subsequent victimization outcomes in adulthood (Gu, Strauss, Bond, & Cavanagh, 2015; Shapiro, Brown, Thoresen, & Plante, 2011), including sexual, physical, and emotional/psychological assault. Findings have revealed alarmingly high rates of early life abuse in later violence-exposed individuals. For example, in a psychiatric sample, women with a history of childhood sexual abuse were found have 3.1 increased odds of experiencing rape or attempted rape after the age of 16 compared to those with no such history (Cloitre, Tardiff, Marzuk, Leon, & Portera, 1996). Furthermore, empirical studies dating back to the 1980s have consistently found a specific connection between women's childhood abuse and later IPV victimization (Briere & Runtz, 1987). Many studies have examined this association, finding expansive evidence that childhood sexual abuse is a risk factor for IPV victimization (Briere & Runtz, 1987; Whitfield et al., 2003). Specifically, Whitfield, Anda, Dube and Felitti (2003) demonstrated that women's childhood physical abuse increased odds of IPV victimization in adulthood by 2-fold and that childhood sexual abuse increased odds by 1.8-fold. Furthermore, Coid and colleagues (Coid et al., 2001), using a convenience sample of 1,207 low-income women recruited during visits to their general practitioners, found that not only is unwanted sexual contact under the age of 16 associated with domestic violence in adulthood, but also that women who had experienced multiple forms of abuse in childhood are at the greatest risk for revictimization as adults. The historical evidence suggests that childhood abuse is an important predictor of IPV victimization.

Research over the past two decades has also found that childhood victimization is a risk factor for experiencing adulthood IPV (Barrios et al., 2015; Berzenski & Yates, 2010; Crawford & Wright, 2007; Daigneault et al., 2009; Fritz, Slep, & O'Leary, 2012; Herrenkohl et al., 2004;

Iverson, McLaughlin, Adair, & Monson, 2014; Kim, 2017; Kimerling, Alvarez, Pavao, Kaminski, & Baumrind, 2007; Ouellet-Morin et al., 2015; Palazzolo, Roberto, & Babin, 2010; Palmetto, Davidson, Breitbart, & Rickert, 2013; Trickett, Noll, & Putnam, 2011; Widom, Czaja, & Dutton, 2014). Using data from 11,056 women from the California Women's Health Survey, Kimerling and colleagues (2007) revealed that approximately 50% of women with a history of childhood violence experience some sort of adulthood victimization (not IPV specifically), as compared to 14% of women without this early abuse history. Furthermore, an examination of Peruvian women during early pregnancy (N = 1,521) indicated that women with a history of childhood physical and sexual abuse had a 7.14-fold increase in lifetime odds of physical and sexual IPV (Barrios et al., 2015). Findings from these studies consistently confirm high rates of IPV in women with histories of childhood abuse. However, some of this research does not differentiate the type of childhood violence experienced (i.e sexual, physical, and/or emotional; Kimerling et al., 2007; Ouellet-Morin et al., 2015), inhibiting a nuanced understanding of the type of abuse associated with this risk.

Other empirical work has specifically examined the early experience of childhood sexual abuse on future abuse risk, finding a strong link with adulthood IPV (Alexander, 2009; Barrios et al., 2015; Becker, Stuewig, & McCloskey, 2010; Berzenski & Yates, 2010; Crawford & Wright, 2007; Daigneault et al., 2009; Graham-Bermann, Sularz, & Howell, 2011; Jaffe, Cranston, & Shadlow, 2012; Kim, 2017; Stein et al., 2016; Straus et al., 1996). In a sample of Korean immigrant women living in the United States, women with a history of sexual abuse were more than five times more likely to be victims of IPV than those without such abuse history (Kim, 2017). Daigneault and colleagues (2009) revealed that this early victimization was associated with a two to fivefold increased odds of reporting psychological, physical, and sexual IPV

victimization from a current or previous partner, using a sample of women from the Canadian General Social Survey (N=9,170). In contrast to Kim (2017), these findings held even after controlling for age, household income level, high-school diploma, aboriginal status, marital status, country of birth, limitations, ownership of residence, childhood physical assault and their current partners' age, excessive drinking and number of years living together (Daigneault et al., 2009). Furthermore, a history of sexual abuse has been found to be significantly related to sexual IPV victimization (Graham-Bermann et al., 2011; Jaffe et al., 2012). Finally, both Alexander (2009) and Stein and colleagues (2016) independently identified that women's history of childhood sexual abuse was associated with having multiple violent partners across the lifetime. Thus, it appears that childhood sexual assault is not only a risk factor for sexual victimization in adulthood by an intimate partner but may also place women at risk for physical and psychological IPV victimization and for having a greater number of violent partners across the lifetime.

However, an important limitation of this research is the lack of simultaneous examination of childhood sexual abuse (i.e. past occurrence of the trauma) and current PTS symptoms on experiencing IPV with an intimate partner as an adult (Alexander, 2009; Barrios et al., 2015; Berzenski & Yates, 2010; Crawford & Wright, 2007; Daigneault et al., 2009; Jaffe et al., 2012; Kim, 2017). Given that PTS in adulthood has been linked to experiencing abuse in childhood (Cloitre et al., 2009), it is important to simultaneously delineate whether it was the occurrence of the abuse or some of the ways that the abuse is currently affecting a person's functioning through PTS symptoms that contribute risk for adult IPV victimization. Continued longitudinal empirical research is needed that examines both history of child sexual abuse and current PTS symptoms to delineate the differential contributions to IPV victimization risk in adulthood.

Prior IPV Victimization as a Risk Factor for IPV Victimization

Extant evidence consistently shows that prior physical and emotional IPV victimization is a risk factor for experiencing future IPV (Kuijpers et al., 2011; Kuijpers, Van der Knaap, & Winkel, 2012). For example, several studies have identified that severity (Krause et al., 2006; Perez & Johnson, 2008) and frequency (Hirschel & Hutchison, 2003; Sonis & Langer, 2008) of prior physical victimization is associated with future IPV victimization. In more recent work, Jouriles, Choie, Rancher, and Temple (2017) followed 843 adolescents over five years and found that prior teen dating violence contributed significant risk for physical adulthood IPV victimization. A meta-analysis of 92 studies of predictors of perpetration and victimization further showed that IPV before pregnancy was a strong risk factor for future IPV during pregnancy (James et al., 2013). Stein and colleagues (2016) also found that current psychological but not physical IPV was associated with revictimization, specifically with having a greater number of violent partners. Furthermore, frequency of current sexual violence was negatively related to having fewer violent partners, although this study was limited by the use of cross-sectional data, which precludes drawing conclusions about the temporal nature of these relationships.

A major limitation of the empirical evidence on prior IPV as a predictor of future IPV victimization is the large variability in definitions and operationalizations of both prior and future IPV victimization across studies (Kuijpers et al., 2011). While some work considers IPV victimization to encompass two or more subtypes of IPV (e.g. physical, emotional, and/or sexual IPV; Kuijpers et al., 2011), other studies only consider physical IPV (Jouriles et al., 2017). Further still, other research conceptualizes revictimization as any future victimization to IPV with any partner, while other work is explicitly concerned with IPV victimization with new or

different violent intimate partners (Ørke et al., 2018). This body of empirical evidence has been used to make generalized conclusions about the potential risk for IPV despite reliance on different constructs of interest across studies both as predictors and outcomes. Continued longitudinal research is needed that isolates the different subtypes of IPV (e.g. physical, emotional, and sexual) as predictors and outcomes and specifically examine risk for IPV victimization across multiple violent intimate partners.

Cumulative Trauma

Over the past three decades, significant attention has been given to the exploration and understanding of the cumulative impact of trauma on health and well-being outcomes. In particular, findings from the groundbreaking Adverse Childhood Experiences (ACE) Study (Felitti et al., 1998) revealed that victimization to four or more categories of childhood adverse experiences was associated with a 4- to 12-fold increase in significant and potentially fatal negative health outcomes compared to individuals who had no victimization. Furthermore, using the same data, Whitfield, Anda, Dube, and Felitti (2003) revealed that each of the examined adverse childhood traumatic experiences reviewed increased IPV victimization risk by approximately two-fold. The authors concluded that there is a graded relationship between the number of traumatic experiences in childhood and IPV victimization risk. Other empirical work has confirmed the graded effect of cumulative childhood trauma on future victimization risk (Jankowski et al., 2002). Additional research identified that the odds of having a new violent partner was predicted by a cumulative lifetime victimization index (Cole et al., 2008), which included multiple types of interpersonal traumas (e.g. IPV and non-partner violence) and gave more weight to childhood experiences than those in adulthood. Longitudinal research is needed

to continue to understand the impact of past interpersonal trauma on risk for experiencing IPV victimization in adulthood.

The next sections reviews the literature on sociodemographic factors as contributors to risk for experiencing IPV victimization. These include race and socioeconomic status.

Sociodemographic Factors and Relationship to IPV

Race

Although significant attention has been paid to understanding the relationship between race and IPV victimization risk in women, the findings have largely remained inconclusive (Caetano, Schafer, & Cunradi, 2001; Cunradi, Caetano, & Schafer, 2002; Kantor, Jasinski, & Aldarondo, 1994). Some evidence indicates that Latina and Black women experience partner violence at higher rates than their White peers, with results showing that the rate of reoccurrence of severe IPV in Latina and Black women is four and six times higher, respectively, than the rate for Whites (Caetano, Field, et al., 2005). Further, approximately 46% of Alaskan Indian and Alaska Native women experience physical violence by an intimate partner in their lifetimes, which is significantly higher than whites or non-Latinas (Breiding, Chen, & Black, 2014). However, other studies have not found significant differences in victimization between the different racial groups (Caetano et al., 2001), making any definitive conclusions about the association of race and IPV impossible.

Research findings on the role of women's race in IPV victimization over the past few decades have remained mixed and the work has primarily focused on three racial categories: White, Black, and Hispanic/Latina (Azziz-Baumgartner, McKeown, Melvin, Dang, & Reed, 2011; Bonomi, Anderson, Cannon, Slesnick, & Rodriguez, 2009; Breiding, Black, & Ryan, 2008; Cho, 2012; Clark, Galano, Grogan-Kaylor, Montalvo-Liendo, & Graham-Bermann, 2016;

Golden, Perreira, & Durrance, 2013; Lipsky, Caetano, & Roy-Byrne, 2009; Miller-Graff & Graham-Bermann, 2016; Nowotny & Graves, 2013; Stein et al., 2016; Walton et al., 2009). On one hand, Miller-Graff and Graham-Bermann (2016) revealed that minority women experienced more physical IPV and higher frequency of violence related to injury compared to non-minority peers. Golden and colleagues (2013) also found that the prevalence of any IPV was highest among Hispanics compared to non-Hispanic White, non-Hispanic Black, and non-Hispanic other. However, in other work, Black women and Latinas had IPV-related police report rates that were two to three times higher than White women (Lipsky et al., 2009) and were more likely to be killed by an intimate partner than White women (Azziz-Baumgartner et al., 2011).

Some scholars have tried to explain the contradictory findings, suggesting that socioeconomic status may be driving this association because race and socioeconomic status are correlated. To rule out findings that confound IPV with poverty, socioeconomic status must be included as a control variable due to the greater proportion of racial and ethnic minority women who live in poverty (Williams et al., 2016). When controlling for socioeconomic status, Latinas (Field & Caetano, 2003) and Latina and Black women (Caetano et al., 2005) were found to be three times more likely to experience IPV than their White counterparts. However, Clark and colleagues (2016) showed that while Latinas had higher levels of violence compared to white peers, Black women did not, when controlling for income, level of education, employment status, shelter residence, relationship status, general health, housing instability, and partner residence. In contrast, when examining the issue of chronicity of IPV, Stein and colleagues (2016) revealed that White and Black women reported having significantly more violent intimate partners than Latinas even after controlling for socioeconomic status. In other work yet, multiracial women were significantly more likely to experience lifetime IPV than non-Hispanic

White counterparts (Breiding et al., 2008). Finally, Bonomi and colleagues (2009) found that Latinas and non-Latinas have comparable prevalence rates of lifetime IPV victimization when income was included as a covariate.

Even after controlling for socioeconomic status, the empirical findings in the extant literature on the role that race may play in IPV victimization risk are not clear. Perhaps, the differential manner of categorizing the racial groups, definitions for IPV, and variation in measurement tools used across studies may account for some of the discrepancies in the findings. For example, in work that utilized face-to-face interviews of study participants (Clark et al., 2016; Miller-Graff & Graham-Bermann, 2016; Stein et al., 2016), IPV victimization tended to be measured using standardized assessment tools (Straus, 1979; Straus, Hamby, Boney-McCoy, & Sugarman, 1996). In contrast, many studies using other interviewing strategies (such as over the phone interviews) did not employ standardized measures of IPV victimization (Breiding et al., 2008; Golden et al., 2013; Nowotny & Graves, 2013). It is possible that this variation in the use of assessment tools between studies may account for some of the inconsistency of findings. Further longitudinal research focused on understanding the link between race and IPV victimization using standardized measures of IPV victimization in women is needed to identify and explicate any potential risk relationship.

Socioeconomic Status as a Risk Factor for IPV Victimization

Empirical research has long found a relationship between lower socioeconomic status and increased risk for IPV (Cunradi et al., 2002; Field & Caetano, 2004; Jewkes, 2002). Families living in economically disadvantaged conditions are more likely to have higher levels of stress, experience greater unemployment, be more socially isolated and have an exacerbated a sense of hopelessness, which may then lead to increased rates of IPV (Cummings, Gonzalez-Guarda, &

Sandoval, 2013; Martin, Tsui, Maitra, & Marinshaw, 1999). More recent empirical work continues to show a link between lower socioeconomic and various forms of victimization, including IPV (L. Davies et al., 2015). For example, using European Union-wide sample of 42,000 women, Reichel (2017) revealed that lower household income was associated with higher rates of IPV. Lower household income was further found to be related to higher IPV in an Australian cohort of 2,041 offspring that participated in a 30-year follow-up study (Ahmadabadi et al., 2017). Low socioeconomic status was found to be a moderate predictor of IPV during pregnancy in a meta-analysis of 92 studies of predictors of perpetration and victimization (James et al., 2013). A clear inverse relationship between income and experiencing IPV victimization has been identified in the literature, though a limitation of the research is that lower income samples have been the predominant population studied.

Major Conclusions and Limitations of Extant Research

The present review of the literature highlights the syndemic relationship between mental health concerns, trauma exposure, sociodemographic factors, and IPV victimization. However, despite the empirical evidence for the overarching contributions of these social epidemics and environmental factors for risk of IPV victimization, much remains underexamined and/or unknown about the nuances of these relationships. For example, as detailed above, there is conflicting evidence for the potential contributing role of PTS, depressive and anxiety symptoms, and alcohol use in IPV victimization risk. In addition, it is unclear if sociodemographic factors such as belonging to a certain racial group may exacerbate IPV victimization vulnerability as a result of socioenvironmental structures.

A major limitation of this body of literature and a possible explanation for the disparate findings on the precise role of these different syndemic predictors on IPV victimization risk are

the inconsistent and, at times, unclear operationalizations of IPV victimization in the research (Cattaneo & Goodman, 2005). The literature is plagued by use of vague terms like “experiencing IPV”, “experiences of IPV”, and “IPV involvement”, lacking specific definitions and operational clarity of the directionality of the IPV (Cattaneo & Goodman, 2005). Furthermore, even when the directionality is clear, many studies only consider a single type of IPV victimization (e.g. physical assault), ignoring other important aspects of the violence such as total IPV victimization, and/or number of violent partners. Despite this inconsistent use in the constructs of interest across the research, significant attention has been given to aggregating the extant findings (Kuijpers et al., 2011). New research is needed that comprehensively examines the syndemic predictors of mental health and trauma exposure to all types of IPV victimization (total amount of IPV victimization, number of violent partners (IPV re-engagement), and IPV victimization vs. no IPV victimization), using clear definitions and operationalizations of the violence constructs.

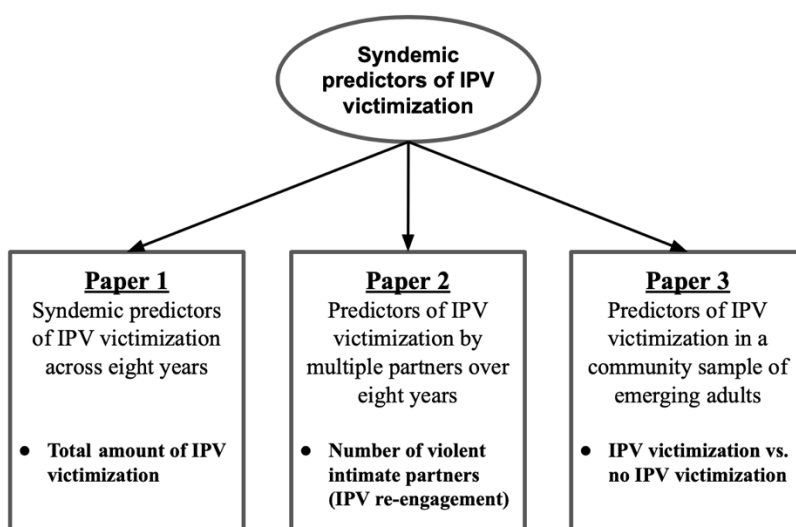
The existing literature is further limited by the overall siloed approach to conceptualizing factors of risk for IPV victimization. Studies often consider the contributing role of mental health concerns or trauma exposure, but rarely consider both, missing the interacting and additive nature of these overlapping social epidemics (Dutton, 2009). Research is needed that utilizes a syndemics approach to the three epidemics of mental health concerns, trauma exposure, and IPV victimization to more comprehensively delineate IPV victimization risk. This framework for studying this vulnerability is advantageous as it further allows for the consideration of the role sociodemographic factors on exacerbating risk. Future research using a syndemics perspective is needed to comprehensively capture the intricate and complex nature of IPV victimization risk. Furthermore, given the high rates of bidirectional violence, continued work would additionally

benefit from using this same framework to understand if the syndemic variables of mental health and trauma exposure differentially contribute risk to experiencing IPV victimization (yes/no).

The Dissertation Studies

This dissertation aims to address several of the identified gaps in the literature as well as extend the current understanding of IPV victimization risk using a syndemics framework in three independent empirical studies. A clear understanding of the contributing mechanisms of vulnerability for IPV victimization will inform the refinement and development of prevention programs and treatment interventions that specifically target these risk factors as a pathway to effective and sustained IPV victimization reduction and mitigation.

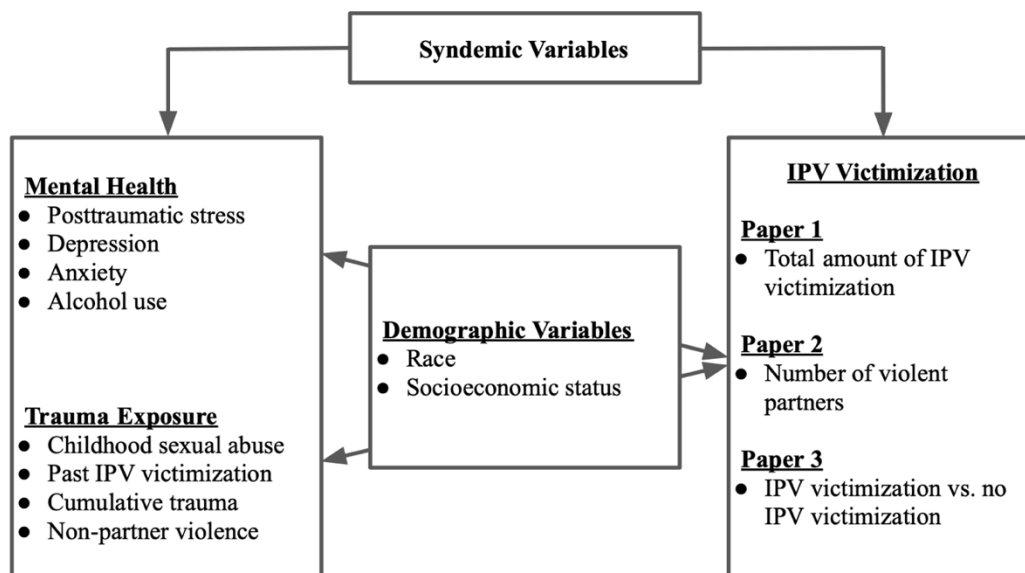
Figure 1.2. Conceptual flow of the three dissertation studies



The dissertation will focus on three unique aspects of IPV victimization. The first study will examine the syndemic predictors of mental health, trauma exposure, and sociodemographic factors for total amount of IPV victimization over eight years. The second study will delineate the syndemic predictors of mental health, trauma exposure, and sociodemographic factors on intimate partner violence victimization by multiple partners over eight years. The third paper will examine the syndemic predictors of experiencing IPV victimization versus no IPV victimization

in a community sample of emerging adults (See figures 1.2 and 1.3 for a conceptual map of the three dissertation studies).

Figure 1.3. Dissertation Conceptual Model



The overarching aims of the dissertation are as follows:

Aim 1. To identify the contributions of trauma exposure, mental health, and sociodemographic factors to total amount of IPV victimization in women with children over an eight-year period.

Aim 2. To prospectively examine the trauma exposure, mental health, and sociodemographic factors that contribute risk for experiencing IPV re-engagement in women with children across eight years.

Aim 3. To delineate the independent and cumulative roles of trauma exposure and mental health concerns on odds of experiencing IPV victimization in a community sample of emerging adults recruited from an urban emergency department.

Chapter II

The Contributions of the Posttraumatic Stress Symptom Domains to Intimate Partner Violence Victimization Across Eight Years

Intimate partner violence (IPV) victimization is a pervasive public health problem with severe long-term physical and mental health consequences (Bonomi et al., 2006; Smith et al., 2018; Spencer et al., 2019). The financial costs of IPV are high with an estimated population economic burden of \$3.6 trillion, 59% of which are direct medical costs (Peterson et al., 2018). Although decades of political activism and research have brought important attention to this pervasive social problem, empirical work has yet to be able to clearly delineate the core mechanisms that confer risk for IPV victimization in women (Spencer et al., 2019; Stith et al., 2004). Yet, this is an area of urgent need as current treatments for IPV victimization have shown limited effectiveness at reducing future victimization, especially over several years (Eckhardt et al., 2013), further highlighting how current mechanisms of risk targeted through these intervention programs are lacking precision for sustained IPV mitigation over time. The lack of clear indicators of risk may be partially attributable to a dearth of prospective, longitudinal data on women's IPV victimization over multiple years. This study follows women with IPV victimization who have children across eight years to identify the trauma exposure, mental health, and sociodemographic mechanisms of risk for IPV victimization and will extend the current literature by providing a more precise delineation of the confluence of factors that create risk for experiencing IPV victimization in order to inform novel targets of intervention.

The theory of syndemics or synergistic epidemics (Singer, 2009) offers a comprehensive framework for delineating the complexity of the potential mechanisms of IPV victimization risk. Syndemics purports that diseases or epidemics overlap and mutually contribute to one another, resulting in both the clustering of and interaction between said epidemics (Singer, 2009; Singer, Bulled, Ostrach, & Mendenhall, 2017; Singer & Clair, 2003). Central to the theory is the idea that the social contexts in which these interactions occur fundamentally contribute and exacerbate how the epidemics interact. Human social contexts or environments are comprised of dominant structures of social relationships, such as social inequality and injustice (e.g. financial resources), which profoundly contribute to and aggravate the clustering and overlap of social problems (Singer, 2009; Singer, Bulled, Ostrach, & Mendenhall, 2017). Decades of empirical work has shown a consistent overlap in three epidemics: IPV victimization, trauma exposure (e.g. IPV re-engagement and cumulative trauma exposure; Daigneault, Hébert, & McDuff, 2009; Whitfield, Anda, Dube, & Felitti, 2003) and mental health concerns (e.g. traumatic stress and depression; Ehrensaft et al., 2006; Spencer et al., 2019), suggesting a syndemic relationship between these three social concerns. Furthermore, social contextual factors (e.g. income and age) have been linked to differing rates of all three epidemics and severity outcomes (Golden et al., 2013; James et al., 2013; Lipsky et al., 2009). Although some empirical work has individually examined trauma exposure (Whitfield et al., 2003), mental health (Kuijpers, van der Knaap, et al., 2012), or sociodemographic factors (Miller-Graff & Graham-Bermann, 2016) as indicators of IPV victimization risk, limited research has concurrently examined these factors as overlapping epidemics. A clear delineation of these contributing risk mechanisms for IPV victimization could inform novel targets of intervention to improve the precision of treatments.

Trauma exposure and IPV victimization risk

IPV re-engagement. Trauma exposure is a known risk factor for IPV victimization (Whitfield et al., 2003). IPV re-engagement or ‘re-engagement is the number of distinct violent partners that a person has over a specific period of time regardless of the amount of IPV victimization with any specific partner. Although a newer area of inquiry, IPV re-engagement has been preliminarily associated with specific types of IPV victimization across time, particularly more psychological and less sexual IPV victimization (Cole et al., 2008; Stein et al., Under Review, 2019). Continued longitudinal work is needed to determine if women’s IPV re-engagement confers risk for greater total IPV victimization across time.

Cumulative trauma. Robust empirical work has demonstrated the graded relationship between the amount of adverse childhood events experienced and IPV victimization risk (Barrios et al., 2015; Cole et al., 2008; Jankowski et al., 2002; S. F. Stein et al., 2019; Whitfield et al., 2003) For example, in the seminal work, Whitfield, Anda, Dube, and Felitti (2003) showed that each Adverse Childhood Experience (ACE) during childhood increased IPV victimization risk in adulthood by approximately two-fold. Daigneault and colleagues (2009) revealed that this early victimization was associated with a two to fivefold increased odds of reporting psychological, physical, and sexual IPV victimization from a current or previous partner, using a sample of women from the Canadian General Social Survey (N=9,170). Despite these robust findings, limited work has examined the contributions of cumulative trauma over the whole lifespan to IPV victimization risk (Smith & Stover, 2016).

Mental health and IPV victimization risk

Posttraumatic stress. Mental health concerns are a known risk factor for IPV victimization (Ehrensaft et al., 2006; Spencer et al., 2019). For example, posttraumatic stress disorder (PTSD) or posttraumatic stress symptoms (PTS), a concern associated with the

dysregulation of the nervous system that may occur following exposure to a traumatic event or events (A. N. Schore, 2002), have been consistently linked with IPV victimization (Iverson et al., 2011; Krause et al., 2006; Messing et al., 2012; Perez & Johnson, 2008; Smith & Stover, 2016; Spencer et al., 2019) and chronic IPV victimization, such as IPV re-engagement (Stein et al., Under Review). Posttraumatic stress may compromise an individual's ability to accurately read, assess, and respond to environmental danger cues using present-moment data (Iverson et al., 2011; Van der Kolk, 1994, 2015). In the context of IPV, PTS may impede the capacity to correctly and effectively attend and respond to signs of threat from an intimate partner (Iverson et al., 2011). However, other studies have not found a relationship between PTS and IPV victimization risk (Cole et al., 2008; Sonis, 2008; S. F. Stein et al., 2019). Continued prospective work using longitudinal data is needed to further delineate the contributions of PTS to IPV victimization.

The inconsistencies in these findings may point to the potential limited clinical utility of using PTS as a total construct when examining mechanisms of risk. PTS is characterized by diverse symptom presentations (Foa et al., 1995; Schauer & Elbert, 2010; Van der Kolk, 1994), comprised of constellations of the PTSD symptoms domains of reexperiencing (e.g. somatic or image-based flashbacks), avoidance (e.g. avoidance of distressing memories, thoughts, feelings, or external reminders of the traumatic event), negative alternations in cognitions and mood (e.g. feeling isolated), and arousal (e.g. hypervigilance; American Psychiatric Association, 2013). For example, dissociation and hyperarousal are characteristics of PTS but are physiologically and experientially very different and may differentially contribute risk for IPV victimization. Dissociation is associated with parasympathetic nervous system activation and escaping present moment events whereas hyperarousal is connected to sympathetic arousal and potentially hyper-

attunement to present moment stimuli (Schauer & Elbert, 2010). Collapsing these disparate symptoms into a single construct may be too imprecise to be able to identify the exact mechanisms of risk for IPV victimization.

However, very limited work has examined the contributions of the specific PTS symptom domains on IPV victimization (Dutton, 2009; Iverson et al., 2013; Krause, Kaltman, Goodman, & Dutton, 2006). In a 6-month longitudinal study, hyperarousal symptoms but not numbing, avoidance, or reexperiencing predicted experiencing further physical IPV in women (Iverson et al., 2013). In contrast, another study found that PTS reexperiencing was associated with increased physical and psychological IPV over a 6-month time period (Kuijpers, van der Knaap, et al., 2012). Yet another investigation identified that PTS numbing increased women's odds of experiencing further IPV by 1.49 times over a year period, which equates to a three-fold increase in IPV victimization odds when shifting from endorsing no numbing symptoms to two (Krause et al., 2006). In further work, avoidance/numbing, reexperiencing, nor arousal predicted future IPV victimization (Cogle et al., 2009). Despite a prior call for research to delineate the potential contributions of the PTS symptom domains on IPV victimization risk (Dutton, 2009), empirical work has been limited with overall inconclusive findings.

Depression. Some empirical work has established a link between depression and future IPV victimization (Caetano et al., 2008; Cogle et al., 2009; Iverson et al., 2011; Lehrer et al., 2006; Spencer et al., 2019). In a national probability sample of 2,863 women, major depression was found to increase the odds of new IPV victimization by 1.69 times over two years (Cogle et al., 2009). Iverson and colleagues (2011) found that reductions in depression were significantly associated with lower levels of physical IPV victimization over 6-months. Still, other studies have failed to find that depression is predictive of IPV victimization (Cole et al., 2008; Perez &

Johnson, 2008; Renner & Whitney, 2012; Sonis & Langer, 2008; S. F. Stein et al., 2019; S.F. Stein et al., n.d.). For example, depression in the two weeks prior to baseline was not associated with experiencing IPV with a new partner over the subsequent year in a study of 756 women who had obtained a domestic violence order against an intimate partner (Cole et al., 2008). Continued research using prospective longitudinal data over a longer period of time is needed to delineate any role of depression in creating risk for IPV victimization.

Sociodemographic factors and IPV victimization risk

Sociodemographic factors including household income and age have also been associated with IPV victimization risk (Miller-Graff & Graham-Bermann, 2016; Spencer et al., 2019). Lower socioeconomic status has long been identified as a risk factor for IPV victimization (Cunradi et al., 2002; Field & Caetano, 2004; Jewkes, 2002; Spencer et al., 2019). Families living in economically disadvantaged conditions are more likely to have fewer economic resources to get out of difficult situations, higher levels of stress, experience greater unemployment, be more socially isolated and have an exacerbated a sense of hopelessness, which may then lead to increased rates of IPV (Cummings et al., 2013; L. Davies et al., 2015; S.L. Martin et al., 1999). Using European Union-wide sample of 42,000 women, Reichel (2017) found that lower household income was associated with higher rates of IPV. Lower household income was further found to be related to higher IPV in an Australian cohort of 2,041 offspring that participated in a 30-year follow-up study (Ahmadabadi et al., 2017). In addition, women's age has been shown to be a strong indicator of IPV victimization such that older women have less IPV victimization than their younger counterparts, though these effects are small (Spencer et al., 2019). Findings suggest that income and age are integral considerations when examining IPV victimization risk.

Study aims and hypotheses

The present study follows 118 women with children over eight-years who have experienced IPV victimization to prospectively examine the factors that may confer risk for IPV victimization. The aim of this study is to identify the respective contributions of trauma exposure, mental health, and sociodemographics to IPV victimization in order to inform novel targets of intervention to improve the precision of current treatments. Given the review of the literature, the following relationships are anticipated: 1) Total amount of women's PTS symptoms will be positively associated with IPV victimization. 2) Income will be inversely associated with IPV victimization such that women with lower levels of income will experience more IPV victimization over time. 3) Women's age will be inversely associated with IPV victimization such that younger women will experience greater IPV victimization. Findings for the relationship between IPV re-engagement, cumulative trauma, depressive symptoms, and PTS symptom domains are inconclusive and thus the expected directions of these associations are unknown. This study addresses the exploratory research questions: 1) Is IPV re-engagement associated with amount of IPV victimization across time? 2) Is lifetime cumulative trauma exposure related to women's IPV victimization over time? 3) Do women's depressive symptoms across eight years contribute risk for IPV victimization? 4) Which of PTS symptom domains contribute risk for total IPV victimization over time?

Method

Participants

One hundred and eighteen women with children participated in a randomized control trial evaluation of a treatment program indicated for mothers who had experienced IPV victimization (Graham-Bermann, 2011). At baseline, women were 21 to 54 years of age ($M=31.79$, $SD=7.23$)

from different ethno-racial groups: 48.31% White, 36.44% Black, 5.93% Latina, and 9.32% other. This is a low-income sample with a mean monthly household income of \$1,338 (SD=\$1,386) and 41% of women reported having only completed high school or less.

Participant enrollment in the study started in 2006. Women who had experienced violence by an intimate partner within the prior two years and who had a child in the target age range (4-6 years) were eligible for participation in the trial and were randomly assigned to either the intervention (receipt of the intervention) or a control condition. Fifty-nine women (49.17%) were assigned to the treatment group. Participants completed four clinical interviews over an eight-year period: at baseline (time 1), 5-weeks after enrollment (time 2), 6-8 months after enrollment (time 3), and 8-years after enrollment (time 4). This study utilizes data from waves 1, 3, and 4, as IPV victimization was not assessed at wave 2.

Procedures

Following approval from the university's Institutional Review Board, participants were recruited using flyers and pamphlets posted in local businesses, school campuses and social service agencies in Southeast Michigan and Southern Ontario, Canada. Referrals for study participation were also given from local agencies providing services to women who had experienced IPV victimization. One hundred and fifty women expressed interest in participating in the study: of this group, 25 did not meet inclusion criteria and five decided not to participate due to scheduling difficulties. Sequential assignment using block randomization was used to assign the remaining 118 participants to either the treatment (intervention) or the control (no intervention) condition. Fifty-seven women were assigned to the intervention group.

The Moms' Empowerment Program (MEP; Graham-Bermann, 2011) is a manualized group intervention with two meetings per week for five weeks and with six to eight women per

group. Therapists were from local mental health clinics and advocacy agencies, or graduate students in clinical psychology and social work. All group leaders completed a six-hour training in implementing the program. A licensed clinical psychologist provided weekly group supervision to ensure adherence to the intervention protocol.

All women completed a structured clinical interview (time 1) after providing written informed consent. Female research assistants and graduate students in clinical psychology and social work trained in clinical interviewing techniques and research ethics administered the interviews and were supervised by a licensed psychologist. Participants responded to questions about IPV victimization, mental health, trauma history, and sociodemographic information. Interviews lasted approximately one to two hours and participants were compensated in the amount of \$20 dollars for their time for the first interview. All rights of the participants were protected. Following the completion of the intervention program or the five-week waiting period, participants completed a second clinical interview (time 2). A second follow-up evaluation to assess continued progress of study variables of interest was administered 6-9 months later (time 3).

The present study added an eight-year follow-up assessment (time 4). Initial study contact for time 4 requirement relied on contact information provided by participants at the prior measurement occasions. If women were unable to be reached using this information, subsequent recruitment procedures were implemented including using social media (Facebook, Google +, and Instagram) and online searches and people finding search engines. Information included in the follow-up recruitment attempts was intentionally vague to protect the privacy and safety of the women. The Institutional Review Board approved all study procedures and materials.

Upon contact with study staff, women were provided an explanation of the fourth study phase and their participation was requested. Sixty-eight women were located and agreed to the follow-up clinical interview. Similar procedures to the first three time points were followed for the interview process. Women were given the option to be interviewed at their residence if they deemed it appropriate and safe, or provided options to meet at local public places such as libraries and local business. Three of the participants no longer lived in Michigan; study staff travelled to their new states of residence to complete in-person clinical interviews. Women were compensated in the amount of \$75 in cash for their participation in this phase of the study.

Measures

IPV victimization. The Revised Conflict Tactics Scale (CTS-2; Straus, Hamby, Boney-McCoy, & Sugarman, 1996) was used to assess women's victimization to IPV. The full CTS-2 measures both perpetration of and victimization to physical assault, sexual coercion, psychological aggression, injury, and negotiation with an intimate partner but due to the aims of the study only the victimization items were administered to participants. Respondents reported on their frequency of victimization to 39 different types of IPV events on a seven-point Likert ranging from 0 (never) to 6 (20 times or more). The measure includes questions such as, "My partner slapped me" or "My partner used force to make me have oral or anal sex". To more accurately assess the frequency of IPV victimization, item responses were recoded to represent the midpoint of each response category (e.g., 6 to 10 times was recoded as 7.5). A mean total IPV score was created by summing all subscales with the exception of the negotiation scale (Straus et al., 1996). Reliability for the total IPV was (α) .92.

IPV re-engagement. IPV re-engagement was measured at each measurement occasion using a single item: "How many violent partners have you had in your lifetime?". Given that the

question asks about lifetime IPV re-engagement, the count should either remain the same or increase across the measurement occasions. However, at time 3, eight women reported fewer violent partners than at time 1, and at time 4, 13 women reported having fewer partners than they did either time 1 or time 3. To address this discrepancy, if women reported fewer violent partners at time 3 than time 1, their time 1 count was used to replace their original time 3 count. If their time 4 count was lower than either their time 1 or 3 count, the higher value of the two time points was used to replace the originally reported time 4 count.

Cumulative trauma. A score of participant's reported cumulative history of exposure traumatic events was evaluated using items from the PDS (Edna B. Foa et al., 1997). Respondents either endorsed or denied experiencing 12 traumatic events including nonsexual and sexual assault, childhood sexual abuse, torture, and having been held hostage. nonsexual and sexual assault, military combat or a war, imprisonment (for example, prison inmate, prisoner of war, or having been held hostage), and torture. At baseline, women were asked about lifetime history, whereas at the subsequent measurement occasions the timeframe of the items was since the last interview. An initial sum score was created of the endorsed 12 trauma items at baseline. The cumulative trauma score at time 3 was calculated by summing the baseline score with the added trauma reported since baseline. The cumulative trauma score at time 4 was calculated by summing the baseline interpersonal score, the added trauma reported between baseline and time 3, and the added trauma reported between time 3 and time 4.

Posttraumatic stress. Women's PTS at each time point were assessed using the Posttraumatic Diagnostic Scale (PDS; Foa, Cashman, Jaycox, & Perry, 1997), which measures the frequency of PTS over the previous month using a Likert scale from 0 (not at all or only one time) to 3 (5 or more times a week/almost always). Sample items included, "experiencing

physical reactions when reminded of the traumatic event” and “being jumpy or easily startled”. A mean sum score of PTS was calculated with a possible score range from 0 to 51. Sum scores above 23 have been found to indicate significant posttraumatic stress symptomatology and represent a likely diagnosis of PTSD (Sheeran & Zimmerman, 2002), although this score in isolation is insufficient to determine whether DSM-IV diagnostic criteria were met. Mean sum scores for the three DSM-IV symptom domains of PTSS (avoidance, reexperiencing, and arousal) were then calculated utilizing the corresponding subscale items (Foa et al., 1997). The PDS has been found to be a valid and reliable measure of posttraumatic stress (Powers et al., 2012). Reliability for the present study was (α) .89. Reliability (α) for the subscales was: .76 for avoidance; .81 for reexperiencing; and .75 for arousal.

Depressive symptoms. Depressive symptoms were assessed at each measurement occasion using the 20-item self-report Center for Epidemiologic Studies Depression scale (CES-D; Radloff, 1977). Women reported on the frequency of experiencing each of the symptoms over the prior week on a 4-point Likert scale ranging from 0 (less than one day per week) to 3 (most or all of the time, 5-7 days). Positive items were reversed coded so that higher values reflected greater frequency of symptoms and a total score was created by summing all items of the scale. The CES-D is not validated for use as a clinical diagnostic instrument, but total scores on above 16 have been found to suggest significant depressive symptomatology (Lewinsohn et al., 1997; Radloff, 1977; Radloff & Teri, 1986). The CES-D is valid and reliable for assessing depressive symptoms (Miller, Anton, & Townson, 2008). Reliability for the present study was (α) .92. Reliability (α) for the subscales was: .82 for depressed affect; .80 for positive affect; .75 for somatic symptoms; .57 for interpersonal (but only has 2 items).

Sociodemographic variables. Respondents provided information on age, racial group of identification, education, employment, and monthly income.

Planned Analyses

Longitudinal multilevel modeling (MLM) was anticipated to be the most appropriate modeling strategy to examine the predictors of risk of IPV victimization over eight years as it is able to account for the high likelihood of correlation between repeated measures in longitudinal data to avoid the underestimation of standard errors (Raudenbush & Bryk, 2002). However, results from an empty model used to calculate the unconditional intra-class correlation coefficient (ICC) indicating the degree to which variation between participants explains variation in the amount of IPV victimization revealed that in this case MLM was not a better approach than ordinary least squares (OLS) regression. Longitudinal OLS regression was therefore used for all subsequent analyses.

Given that data collection spanned eight years, variables at each time point had between 0 - 64% missing data. Missingness in the sample was addressed using multiple imputation (Royston, 2004; Schafer, 1999) based on values of the independent variables included in the statistical model. Twenty imputed datasets were created and then simultaneously analyzed in accordance with standard recommendations (Graham et al., 2007). Regression coefficients and standard errors were then averaged across regression models (Royston, 2004).

First, a model was estimated to determine the effects of the MEP intervention on IPV victimization over the eight years. This model included only time (weeks), intervention group assignment (treatment/control), and the interaction between time and intervention group assignment. The time by intervention assignment group interaction term provides information about variation in time trajectories based on intervention participation.

Predictors of IPV victimization over eight years were next estimated using longitudinal OLS regression. Given the review of the literature, trauma history (IPV re-engagement and cumulative trauma), mental health (PTS and depressive symptoms), and sociodemographic factors (income and age) were included as independent variables. All predictors in the model were time variant with the exception of age. An interaction term between time and total PTS was also included in the model given previous robust findings of the importance of PTS for IPV victimization risk (Spencer et al., 2019). This interaction term provides information about the time trajectory of IPV victimization based on levels of PTS symptoms. A final model was estimated including the previous model predictors and the PTS subscales (instead of PTS total) as predictors of IPV victimization over eight years. All analyses were done using Stata 14.

Results

Means and standard deviations for all study variables at each measurement occasion prior to imputation are summarized in Table 2.1. There were no differences between the treatment and control groups on any of the study's variables of interest at baseline.

Effects of treatment on IPV victimization

To evaluate the effects of treatment on IPV victimization across the eight years, a model was created that included only time, intervention assignment, and the interaction between time and intervention assignment. Time was significantly associated with less IPV victimization ($b = -.21, p < .001$) across the eight years, but intervention assignment was not. The intervention by time interaction coefficient was also not significant ($b = -.02, p = .813$), suggesting no significant differences in total IPV victimization between treatment and control groups across the eight years. As a result, treatment was not included in any subsequent analyses.

IPV victimization risk indicators across eight years

Longitudinal OLS linear regression modeling was used to examine predictors of IPV victimization risk across eight years (see Table 2.2). Initially, women with higher PTS symptoms had significantly higher levels of IPV victimization ($\beta = 0.66, p < .001$). However, this did not hold over time as evidenced by a significant interaction between PTS and time ($\beta = -0.52, p = .029$), suggesting that across time those with higher PTS symptoms decrease more quickly in amount of IPV victimization than those with lower PTS symptoms. Time alone, IPV re-engagement, cumulative trauma, total depressive symptoms, monthly income, and age were not significantly associated with IPV victimization across time.

Posttraumatic stress symptom domain indicators of IPV victimization risk

Next, a separate longitudinal OLS linear regression was used to model the contributions of the symptom domains of PTS on IPV victimization risk, while controlling for all other predictors of risk (See Table 2.2). Initially, women with higher levels of PTS reexperiencing had higher levels of IPV victimization than those with lower levels of PTS reexperiencing symptoms ($\beta = 0.31, p = .027$). The interaction between time and PTS reexperiencing over the eight years was not significant, suggesting that higher levels of PTS reexperiencing remained associated with greater IPV victimization across the eight years. Women with higher levels of PTS arousal at baseline also had higher levels of IPV victimization compared to those with lower levels of PTS arousal ($\beta = 0.28, p = .032$). The interaction between time and PTS arousal revealed a trend level effect ($\beta = -0.36, p = .070$), suggesting that across time those with higher PTS arousal symptoms decrease more quickly in amount of IPV victimization than those with lower PTS symptoms over the eight years. In this model, older women had significantly less IPV victimization compared to younger women ($\beta = -0.16, p = .026$). PTS avoidance symptoms, time

alone, IPV re-engagement, cumulative trauma, total depressive symptoms, and monthly income were all not significantly associated with IPV victimization across time.

Discussion

The aim of the present study was to identify the predictors of trauma exposure, mental health, and sociodemographic factors that may contribute risk for experiencing IPV victimization in women with children. This research is novel in that it follows participants over an eight-year period and utilizes rigorous assessments of IPV victimization, IPV re-engagement, cumulative trauma, PTS, depression, and household monthly income over three measurement occasions collected using clinical interviews. Study results lend mixed support for the proposed hypotheses: Higher levels of PTS symptoms were associated with initially greater IPV victimization. However, across time women with higher PTS symptoms decreased more quickly in amount of IPV victimization than those with lower PTS symptoms. Follow-up analyses revealed that higher levels of PTS arousal and reexperiencing were each associated with initially higher levels of IPV victimization. However, over time women with high levels of PTS arousal decreased more quickly in amount of IPV victimization than those with low levels of PTS arousal. This was not the case for reexperiencing: higher levels of PTS reexperiencing started off and remained associated with higher levels of IPV victimization across time. Finally, older women had significantly less IPV victimization across time. IPV re-engagement, cumulative trauma exposure, depressive symptoms, and monthly income were not associated with IPV victimization across time in this sample.

Trauma exposure, operationalized as IPV re-engagement and lifetime cumulative trauma exposure, was not associated with IPV victimization across the eight years in these women. Although prior work has established a relationship between psychological and sexual IPV

victimization and IPV re-engagement risk, these findings also failed to find a relationship between total IPV victimization and IPV re-engagement risk over time (Stein et al., Under Review, 2019). Furthermore, no prior research has found an association between IPV physical victimization and IPV re-engagement (Alexander, 2009; Stein et al., Under Review, 2019). Current findings suggest that IPV re-engagement is not a risk factor for the total amount of IPV victimization experienced by women with children over eight years. Perhaps IPV re-engagement is an important indicator of risk for certain forms of IPV victimization but not the total spectrum of IPV victimization across time. Continued work is needed to delineate the role of IPV re-engagement in specific forms of IPV victimization risk (including physical, psychological, and sexual IPV) over time.

Lifetime cumulative trauma exposure was also not found to be a significant risk factor for IPV victimization in women with children in this sample across eight years. Prior work has shown a robust and graded association between cumulative trauma in childhood and IPV victimization risk in adulthood (Barrios et al., 2015; Cole et al., 2008; Jankowski et al., 2002; S. F. Stein et al., 2019; Whitfield et al., 2003). However no past research has examined the role that the accumulation of traumatic events from childhood and adulthood may have with regard to predicting IPV victimization risk (Smith & Stover, 2016). Current findings suggest that the collapsing of traumatic events from childhood and adulthood into a single construct is not a useful indicator of risk. Perhaps the collapsing of traumatic events across all of development obscures the measurement of traumatic events during developmentally sensitive periods for brain development and overall functioning. Continued work is needed to delineate the specific types of traumatic events (e.g. interpersonal vs. natural disasters) and the developmentally most sensitive periods that confer most risk for future IPV victimization.

Findings partially supported the hypothesis that total amount of women's PTS symptoms over time would be associated with higher levels of IPV victimization. At baseline, women with higher amount of PTS symptoms had greater IPV victimization compared to those with lower amounts of symptoms. This coincides with past findings that show robust positive associations between PTS and IPV victimization (Iverson et al., 2011; Messing et al., 2012; Spencer et al., 2019; Stein et al., Under Review). However, this did not remain true across time in this sample. Over the eight years, women with higher PTS symptoms decreased more quickly in amount of IPV victimization than those with lower PTS symptoms. Even women with higher levels of PTS symptoms across time seem to be "leveling out" in amount of IPV victimization experienced suggesting that overall total amount of PTS symptoms over time may have less of a central role in IPV victimization risk than current empirical evidence suggests (Spencer et al., 2019). Perhaps, findings from the present study point to the potential limited utility of using PTS as a total construct when examining mechanisms of risk for IPV victimization given that the PTS diagnostic category is comprised of disparate symptoms (Foa et al., 1995; Schauer & Elbert, 2010) that may pose differential risk or protection for IPV victimization. Collapsing these symptoms into a single construct may be too imprecise to be able to identify the exact mechanisms of risk for IPV victimization.

To address the potential limitation of collapsing of the PTS symptom domains into a single construct, a follow up analysis was conducted to determine the role of each PTS symptom domain on IPV victimization risk over the eight years. Findings revealed that that higher levels of PTS arousal and reexperiencing but not avoidance were each associated with initially higher levels of IPV victimization at baseline compared to those with lower levels of arousal and reexperiencing symptoms, respectively. However, over the eight years women with higher levels

of PTS arousal decreased more quickly in amount of IPV victimization than those with lower levels of arousal. This was not the case for reexperiencing: higher levels of PTS reexperiencing started off and remained associated with higher levels of IPV victimization across time. Prior work has also found an association between PTS arousal and physical IPV victimization (Iverson et al., 2013) and PTS reexperiencing and physical and psychological IPV victimization (Kuijpers, van der Knaap, et al., 2012), though Iverson and colleagues (2013) did not find an association between reexperiencing and physical IPV victimization. However, both studies were limited in that they only followed women for a total of six months and utilized statistical methods that did not allow for the examination of the trajectories of PTS symptoms on IPV victimization over time. Findings from the present study suggest that over time PTS reexperiencing not arousal confers significant continued risk for IPV victimization. It may be that reexperiencing symptoms are contributing to continued relational patterning with an intimate partner that elevates risk for interactions characterized by violence. Although continued research is needed to delineate the mechanism by which reexperiencing is leading to increased IPV victimization risk, treatment interventions should begin to address PTS reexperiencing as a key risk factor for IPV victimization across time.

Women's depressive symptoms over eight years were not significantly associated with IPV victimization across time in this sample. This coincides with empirical work that found no significant relationship between depressive symptoms and IPV victimization (Cole et al., 2008; Perez & Johnson, 2008; Renner & Whitney, 2012). However, other evidence suggest a strong association between the two (Iverson et al., 2011; Spencer et al., 2019). The present study differs from other work as it examines IPV victimization across a much longer time period. For example, Iverson and colleagues (2011) found a relationship between decreases in depressive

symptoms and reductions in IPV victimization in women participating in a clinical trial, but only followed participants for six months following the conclusion of the study. Other studies have identified correlations between IPV victimization and depressive symptoms but only use bivariate correlations using cross-sectional data (Cano & Vivian, 2003). Extant evidence may suggest that depressive symptoms may contribute some amount of risk in the short term for IPV victimization but not over a longer period of time. Continued prospective research over many years is needed to continue to discern any potential role that depression may have as a mechanism of risk for IPV victimization over time.

Findings partially support the independent hypotheses that sociodemographic variables of income and age would each be associated with IPV victimization across eight years. Evidence was not found for the inverse relationship between income and IPV victimization in this sample, which stands in contrast to previous work showing robust associations between lower income and higher IPV victimization (Ahmadabadi et al., 2017; Reichel, 2017; Spencer et al., 2019) . However, the present study used a sample of low income women. Findings may just suggest that being at the extremes of the lower income group is not associated with greater IPV victimization risk across time. It may be that all women in this study were under comparable financial strain and thus the data lacked variability to detect any influence on IPV victimization across time. Additional research that includes a sample with greater income level range is needed to more fully discern income's role in IPV victimization across time in women. Study findings from the second model which including the PTS symptom domains did support the hypothesis that women's age would be inversely associated with IPV victimization. In this sample, older women had less IPV victimization over eight years than their younger counterparts, which is in line with prior meta-analytic findings that there is a small effect of age on IPV victimization risk (Spencer

et al., 2019). Older women may have more extensive networks of social support comprised of individuals able to provide more comprehensive support during challenging times than younger women. Interestingly, however, women's age was not a significant predictor of IPV victimization when only accounting for total PTS symptoms over time. These findings suggest that greater characterization of PTS symptomatology allows for a more accurate delineation of developmental effects on IPV victimization obscured when only accounting for total PTS symptoms. Continued work is needed to delineate the mechanisms by which women's age is contributing to increased risk for IPV victimization across time.

Limitations

While this study provides strong initial evidence for the differential role of the PTS symptom domains in conferring risk for women's IPV victimization across eight years, the research is not without limitations. The study was limited by the use of the CTS to measure IPV victimization, which has been criticized for its lack of evaluation of motivating factors for the reported violence (Hamby, 2016; Lehrner & Allen, 2014). Furthermore, following the request of the participating community agencies, items for the evaluation of women's IPV perpetration were not administered. Future research would benefit from utilizing additional standardized measures of IPV that evaluate both distal and proximal contextual factors for the reported IPV and the assessment of both IPV victimization as well as perpetration by women. The addition of further measurement occasions at smaller intervals would also contribute to potential greater reporting precision from the participants for future work.

In addition, the study had limited statistical power due to the sample size which may have affected the ability to detect meaningful differences. Furthermore, due to the highly mobile nature of this high-risk population, there was participant attrition across the four time points,

contributing to concerns of missing data addressed using multiple imputation. Future research should work to increase sample sizes and enhance the recruitment and retention protocols to minimize the loss of participants across the duration of the study.

Conclusions

This study provides a novel examination of women's trauma exposure, mental health, and sociodemographic risk factors for IPV re-victimization using prospective methodology with eight-year longitudinal data. Findings suggest that PTS symptoms are the salient risk factor for IPV victimization. However over time, higher total PTS symptoms did not remain associated with higher levels of IPV victimization. Examination of the PTS symptom domains revealed that reexperiencing was the only domain associated with continued higher levels of IPV victimization in the women in the sample over eight years. This was not the case for either PTS avoidance or arousal, though arousal was associated with initially higher levels of victimization at baseline. Finally, developmental effects were detected on IPV victimization over time only when accounting for the PTS symptom domains. Results suggest that collapsing PTS symptoms into an overall construct may be too imprecise to identify key mechanisms of IPV victimization risk. Intervention efforts should prioritize addressing reexperiencing symptoms as a pathway to limiting future IPV victimization.

Table 2.1.

Descriptive statistics across time

Variable	Time 1 m(SD)/%	Time 3 m(SD)/%	Time 4 m(SD)/%
Age	31.79 (7.23)	-	38.73 (6.72)
Ethno-racial group			
white	48.31%	-	-
Black	36.44%	-	-
Latina	5.93%	-	-
Other	9.32%	-	
Monthly household income	\$1,338 (\$1,386)	\$1,721 (\$1,723)	\$2,706 (\$2,672)
Education: high school or less	40.68%	-	13.43%
Total IPV victimization	190.12 (139.53)	39.37 (58.66)	33.74 (58.49)
IPV re-engagement	1.59 (.97)	1.95 (1.16)	2.01 (1.27)
Cumulative trauma	2.98 (2.04)	4.81 (4.35)	7.93 (5.64)
Total posttraumatic stress	22.00 (11.91)	13.95 (10.71)	17.27 (13.27)
PTS avoidance	8.25 (5.36)	5.62 (5.15)	7.11 (5.94)
PTS reexperiencing	6.23 (4.09)	3.04 (3.31)	3.70 (3.92)
PTS avoidance	7.53 (4.39)	5.16 (4.07)	6.47 (5.03)
Total depressive symptoms	25.66 (13.66)	17.45 (10.54)	19.23 (13.20)

Table 2.2.

Predictors of risk for IPV victimization across eight years.

Fixed Effects	Model 1					Model 2				
	b	β	SE	t	P-value	b	β	SE	t	P-value
Weeks	-0.06	-0.13	0.06	-0.97	.334	-	-	-	-	-
IPV re-engagement	7.80	0.11	5.99	1.30	.197	-	-	-	-	-
Cumulative trauma count	-1.22	-0.09	1.52	-0.80	.425	-	-	-	-	-
PTS Total	4.21	0.66	0.85	4.97	.000	-	-	-	-	-
Weeks * PTS Total	-0.01	-0.52	0.00	-2.24	.029	-	-	-	-	-
Depressive symptoms	1.29	0.22	0.76	1.70	.091	-	-	-	-	-
Weeks * Depressive symptoms	0.00	0.06	0.00	0.26	.797	-	-	-	-	-
Income	0.00	0.00	0.00	-0.02	.981	-	-	-	-	-
Age	-1.65	-0.14	0.93	-1.8	.079	-	-	-	-	-
Weeks	-	-	-	-	-	-0.07	-0.14	0.06	-1.12	.268
IPV re-engagement	-	-	-	-	-	7.59	0.11	5.81	1.31	.195
Cumulative trauma count	-	-	-	-	-	-0.86	-0.07	1.64	-0.53	.602
PTS avoidance	-	-	-	-	-	1.04	0.08	1.66	0.62	.535
PTS reexperiencing	-	-	-	-	-	5.35	0.28	2.46	2.18	.032
Weeks *PTS reexperiencing	-	-	-	-	-	-0.01	-0.12	0.01	-0.65	.520
PTS arousal	-	-	-	-	-	5.99	0.37	2.66	2.25	.028
Weeks * PTS arousal	-	-	-	-	-	-0.02	-0.36	0.01	-1.84	.070
Depressive symptoms	-	-	-	-	-	1.39	0.23	0.75	1.84	.072
Income	-	-	-	-	-	0.00	-0.01	0.00	-0.11	.914
Age	-	-	-	-	-	-1.92	-0.16	0.86	-2.24	.026

Note: β =Standardized beta

Figure 2.1.

Interaction between time (weeks) and total PTS symptoms

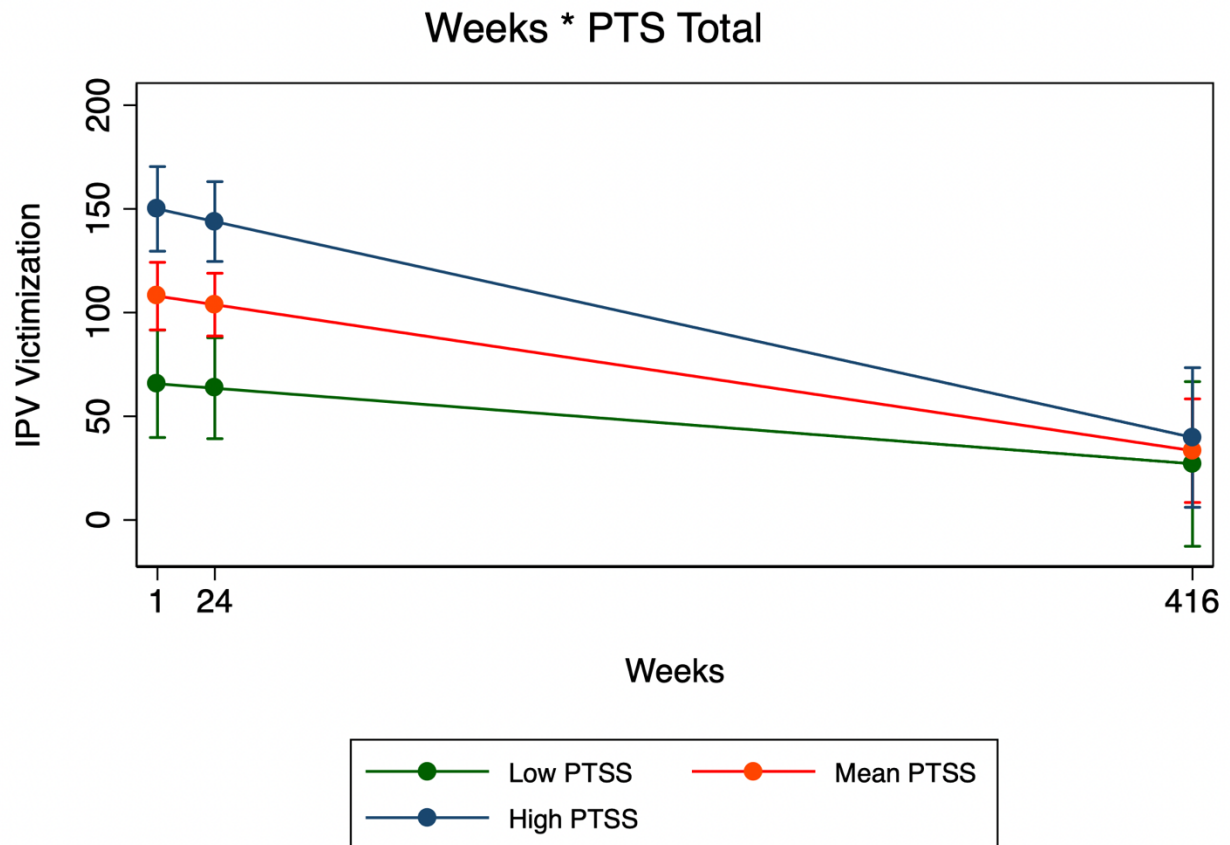


Figure 2.2.

Interaction between time (weeks) and PTS arousal

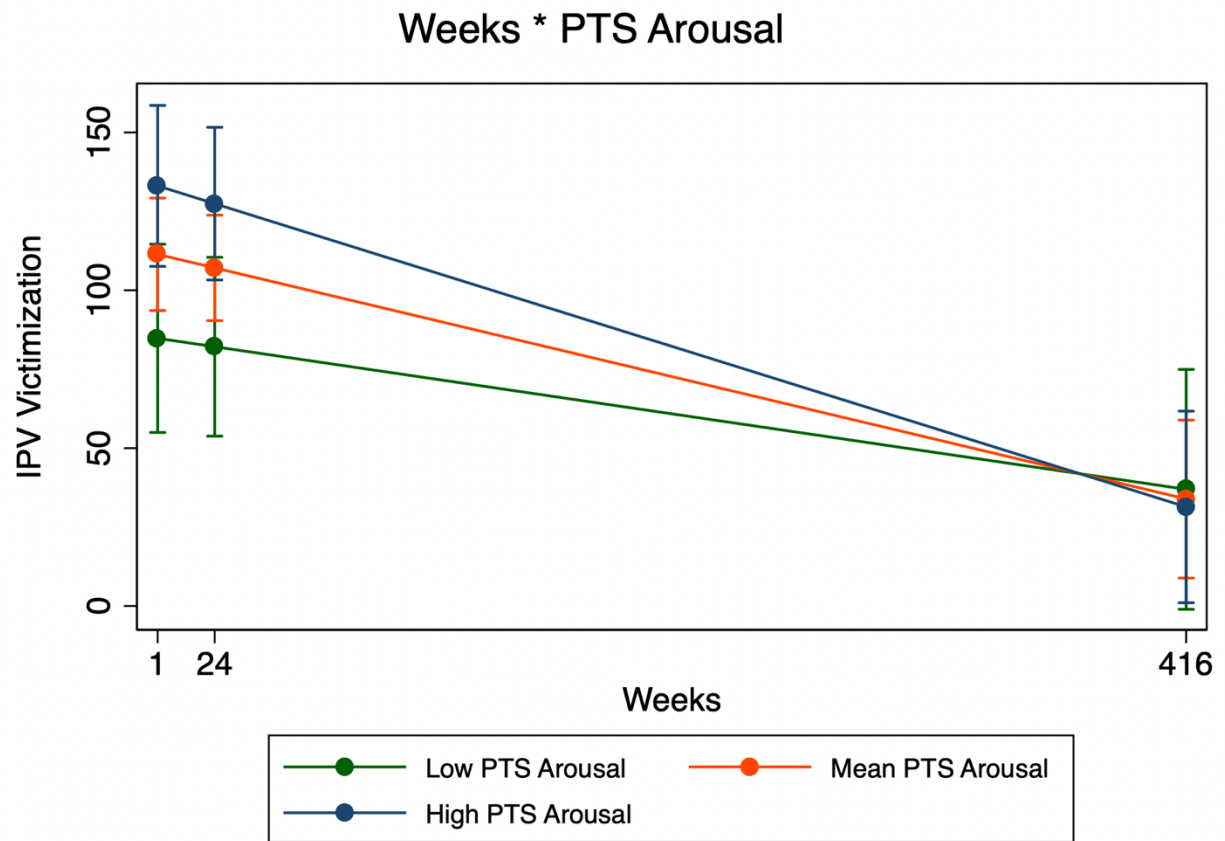
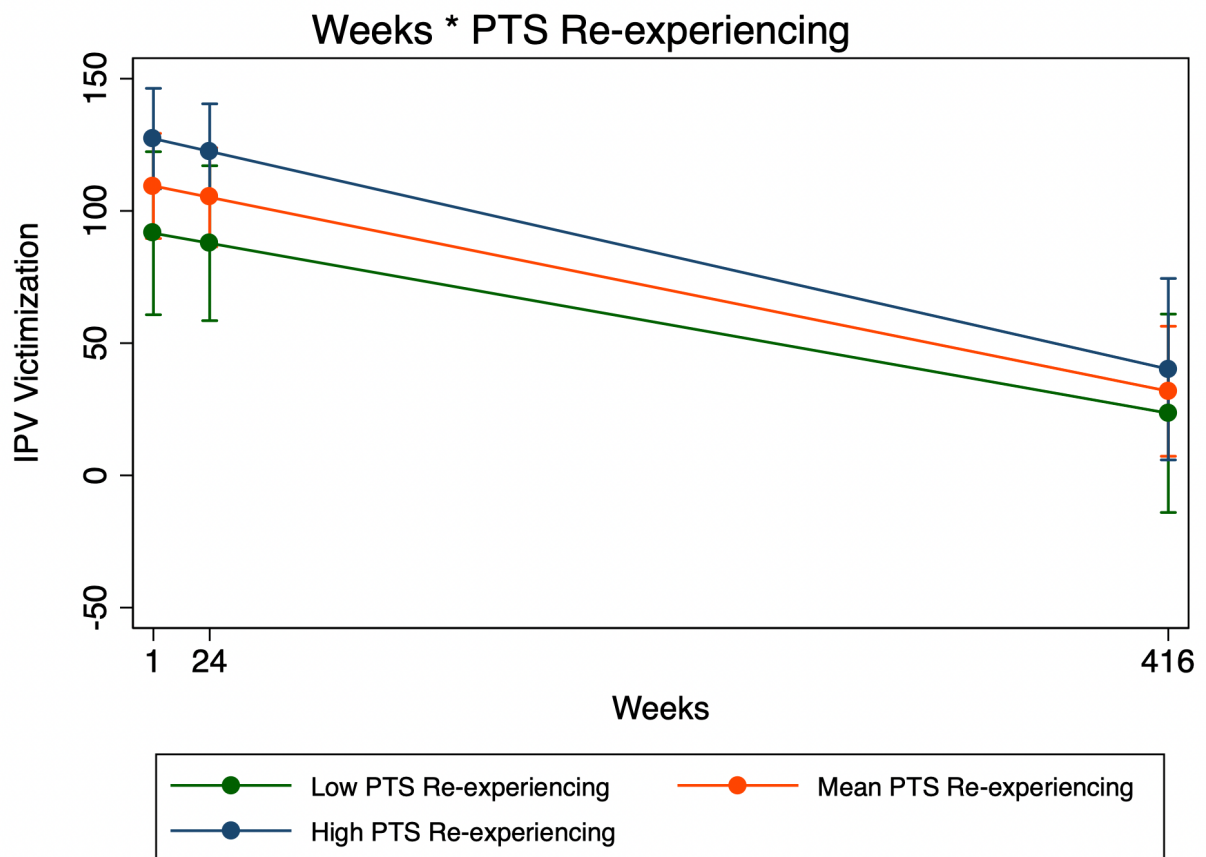


Figure 2.3.

Interaction between time (weeks) and PTS reexperiencing



Chapter III

Predictors of Intimate Partner Violence Victimization by Multiple Partners over Eight Years

Intimate partner violence (IPV) is a pervasive issue that includes acts or threats of physical, sexual, and emotional violence by a current or former intimate partner (Black et al., 2011). Approximately 36% of women experience IPV (S. G. Smith et al., 2018), and its economic toll, including medical, lost productivity, and criminal justice costs, is more than \$100,000 per woman (Peterson et al., 2018). IPV victimization often occurs with more than a single intimate partner; 35 - 56% of women who have experienced IPV report victimization with multiple partners (Alexander, 2009; Stein et al., 2019). IPV victimization that occurs across multiple partners may suggest a chronic pattern of relational disruption. While the reasons for involvement in multiple violent relationships vary, there are likely some individual factors that increase risk. These risk mechanisms are not well articulated (Ørke et al., 2018) despite evidence that having multiple violent partners is associated with poorer outcomes than having a single violent partner (Bogat et al., 2003). Delineating the risk mechanisms of having multiple violent partners can inform efforts to specifically target modifiable risk factors as a pathway to sustained IPV reduction. This study follows women with IPV victimization who have children across eight years to identify the mechanisms of risk for IPV victimization with multiple violent partners.

Evidence suggests that IPV is a dyadic and relational process, meaning that characteristics of both the person perpetrating and the person experiencing the IPV may contribute risk to its occurrence (Kuijpers, et al., 2012b). Theoretical frameworks such as the

Developmental Systems Perspective (Capaldi et al., 2005) also indicate that there may be factors in women's lives that increase their vulnerability to IPV. Although explicating the issue of IPV victimization risk factors has at times been criticized as "victim blaming" (Cattaneo & Goodman, 2005), empirical work has documented the importance of such work in reducing re-victimization (Goodman et al., 2005; Kuijpers et al., 2011) and identifying possible targets of intervention.

Recent work on mechanisms of risk for IPV victimization has focused on risk for having multiple violent partners (Ørke et al., 2018), but this body of research is limited by variability in definitions of IPV victimization across studies (Kuijpers et al., 2011). Terms such as re-abuse, re-victimization, and recidivism are used interchangeably (Cattaneo & Goodman, 2005), and studies vary in whether they define re-victimization as any future IPV, or specifically as IPV with a new partner. Chronic IPV victimization with the same violent partner versus across multiple partners are different phenomena with potentially different etiologies, underscoring the need for clarity of terms and the clear operationalization of constructs.

We use propose the term 'IPV re-engagement' or 're-engagement' to refer to the number of distinct violent partners that a person has over a specific period of time regardless of the amount of IPV victimization with any specific partner. As defined here, the term does not imply or suggest motive for involvement with violent partners, including any indication of 'conscious choosing of a partner due to IPV.' 'IPV re-engagement' was selected over 'IPV revictimization,' coining a new term (re-engagement) that has not yet been used, a contrast to the latter (revictimization), which has long been associated with varying definitions in the literature, as discussed above. The hope is that the introduction of a new, clearly defined term will help to bring greater clarity and precision to the study of this aspect of chronic IPV.

Past trauma victimization as risk for IPV re-engagement

Past IPV victimization has been consistently associated with future IPV victimization risk (Kuijpers et al., 2011; Kuijpers, et al., 2012). One study of 164 treatment-seeking women caregivers who had recently experienced IPV found that more psychological but not physical IPV was associated with re-engagement, whereas more sexual IPV was associated with less re-engagement (Stein et al., 2019). This work is limited by the use of cross-sectional data, which limits conclusions about the temporal order of these relationships. Similarly, although childhood sexual abuse (CSA) has been established a risk factor for IPV victimization and may also confer risk for re-engagement (Alexander, 2009; Barrios et al., 2015; Stein et al., 2019; Vatnar & Bjørkly, 2008), all current work examining the role of child sexual abuse on re-engagement has utilized cross-sectional designs. Longitudinal work from the groundbreaking Adverse Childhood Experiences (ACEs) Study (Felitti et al., 1998) revealed that, in a nationally representative sample of over 8,000 men and women, exposure to each ACE increased risk for IPV in adulthood approximately two-fold (Whitfield et al., 2003), suggesting that there is a graded relationship between traumatic experiences in childhood and IPV risk. However, few longitudinal studies have examined the role of cumulative trauma during both childhood and adulthood, as well as past IPV and sexual abuse, in risk for re-engagement.

Mental health as a mechanism of risk for IPV re-engagement

Posttraumatic stress symptoms (PTS) have been consistently linked to risk for IPV victimization (Iverson et al., 2011; Perez & Johnson, 2008) and are a known outcome of experiencing IPV. However, inconsistent associations have been found between PTS and re-engagement. Some longitudinal evidence has clearly shown that PTS is associated with greater IPV victimization (Blasco-Ros et al., 2010; Iverson et al., 2011), whereas other research has not identified a strong link between PTS and IPV (Cole et al., 2008; Sonis & Langer, 2008; Stein et

al., 2019). The inconsistencies in the findings may be partially accounted for by discrepant conceptualizations of IPV victimization in each study. Moreover, studies varied in whether they examined PTS in relation to re-engagement history versus PTS as a predictor of future re-engagement. Continued investigation using longitudinal data over a longer time is needed to more accurately understand the contributions of PTS to re-engagement.

The inconsistent literature also suggests that the subdomains of PTS may contribute differentially to risk for re-engagement. The presentation of PTSD is nuanced and includes seemingly disparate types of symptomatology (Schauer & Elbert, 2010). For example, numbing and hyperarousal are both symptoms frequently seen in the presentation of PTS but are physiologically and experientially distinct. Numbing includes a potentially blunted responsiveness to the environment whereas hyperarousal may increase sensitivity to trauma-related stimuli. Thus, different PTS symptom domains may differentially contribute risk to re-engagement. However, very few studies have examined the contributions of the specific domains on IPV victimization (Dutton, 2009; K. M. Iverson et al., 2013), and no research to date has examined the role of these domains on IPV re-engagement. Research examining associations between PTS domains and IPV victimization has yielded mixed results, with some studies indicating that numbing increases IPV risk and others suggesting that reexperiencing is more strongly linked to IPV (Cougle, et al., 2009; Iverson et al., 2013; Krause et al., 2006; Kuijpers, et al., 2012a). Despite a prior call for research to understand the contributions of the PTS symptom domains to IPV victimization (Dutton, 2009), evidence remains limited and inconclusive, highlighting the need for further longitudinal research in this area.

Depression has also been examined as a risk factor for future IPV victimization. Prior work has hypothesized that depression may affect the ability to leave a violent situation or a

violent partner due to high levels of guilt and hopelessness and low energy and motivation (Cougle et al., 2009; Iverson et al., 2011). However, the findings are mixed regarding the nature of the relationship between depression and IPV (Iverson et al., 2011; Kuijpers et al., 2011). Although some longitudinal intervention research has established a link between depression and future IPV victimization (Iverson et al., 2011), other work using cross-sectional designs has not (Renner & Whitney, 2012; Stein et al., 2019). Continued prospective longitudinal research is needed to discern the potential role of depression for conferring risk for IPV re-engagement given the mixed nature of extant findings.

Existing literature on IPV victimization risk is limited by the siloed approach to conceptualizing risk factors. Studies often consider the contributing role of mental health or trauma, but rarely consider both, missing the interacting and additive nature of these overlapping social epidemics (Dutton, 2009). Research is needed that concurrently examines the association of past trauma and mental health with re-engagement risk. A framework that examines both ‘what happened to you’ and ‘how you are doing’ – a malleable target of intervention - to understand re-engagement risk is advantageous as it further allows for the consideration of the role of potential sociodemographic factors such as employment, income, and housing instability, previously linked to IPV victimization (Capaldi et al., 2012; Cummings et al., 2013). Identification of the contributing mechanisms for re-engagement will allow for the development of prevention programs and treatments that target these risk factors as a pathway to effective and sustained IPV reduction.

Evidence-based interventions for IPV re-engagement reduction

Interventions indicated for IPV victimization reduction have shown limited effectiveness at reducing future IPV, suggesting the need for continued study of IPV victimization risk

mechanisms and identification of those that are also targetable through treatment (Cattaneo & Goodman, 2005; Eckhardt et al., 2013). Furthermore, to our knowledge, no treatment programs to date have specifically targeted IPV re-engagement mitigation. One exception is the Moms' Empowerment Program (MEP; Graham-Bermann, 2011), a community-based psychotherapeutic intervention for women with children who have experienced IPV. The program includes 10 group sessions designed to reduce IPV victimization (both amount and number of violent partners), traumatic stress, and depressive symptoms. The MEP utilizes an interpersonal perspective, focusing on participants' strengths and capabilities in order to address their biopsychosocial needs. Rooted in empowerment theory, the MEP seeks to address power imbalances in participants' lives as a pathway to making positive changes. Key components of the intervention include creating a sense of empowerment and safety, addressing issues related to the intergenerational transmission of violence, effective communication, emotion regulation, and connecting participants to community resources (Graham-Bermann, 2011). Evidence shows that this program successfully reduced PTS symptoms (Galano et al., 2016; S. A. Graham-Bermann & Miller, 2013), depressive symptoms (Stein et al., 2018), and IPV victimization (L. E. Miller et al., 2014) in White, Black, Latina, and Biracial women. However, continued research is needed to evaluate the effectiveness of the MEP at reducing IPV re-engagement over time.

Aims, hypotheses, and research questions

This study follows women with children who have experienced IPV over an eight-year period to prospectively examine trauma exposure, mental health, and socio-demographic factors (income, employment, and housing instability) and their potential role in creating risk for re-engagement. This study also evaluates the effectiveness of the MEP at mitigating re-engagement over time. The aim of this research is to add to the limited body of knowledge of women with

children's risk factors for IPV re-engagement including trauma exposure, mental health, and socio-demographic indicators. Given the review of the literature, the following relationships are expected: 1) The amount of IPV victimization across eight years will be positively associated with re-engagement; 2) CSA will be associated with greater re-engagement; 3) A greater amount of cumulative interpersonal trauma will be associated with greater re-engagement; 4) Income and employment will be inversely related to re-engagement given the well-established relationship between income and IPV (Capaldi et al., 2012) and 5) Women that participate in the MEP will have lower levels of re-engagement compared to those in the control condition. Findings for the relationship between PTS, depression, and risk for re-engagement are inconclusive and thus the expected direction of this relationship is unclear. This study addresses the exploratory research questions: 1) is PTS over time related to re-engagement? 2) are depressive symptoms over time associated with re-engagement? 3) is housing instability associated with re-engagement?

Method

Participants

Participants were drawn from a RCT of an intervention for mothers who had experienced IPV. Women (N=120) with children who had experienced IPV within the previous two years were randomly assigned to either a treatment or control group, then interviewed at baseline (time 1), 5 weeks (time 2), 6 months (time 3), and 8 years (time 4) after. Fifty-nine women (49.17%) were assigned to the treatment condition (see Figure 1). The study used data from time 1, 3, and 4, as IPV was only assessed at these occasions.

At the time of initial enrollment, women ranged from 21 to 54 years of age ($M = 31.86$, $SD = 7.18$). Participants came from varied racial groups: 47.50% White, 36.67% Black, 5.83% Latina, and 10% other. This was a low-income sample with a mean monthly household income

of \$1,348 (SD = \$1,377; range = \$0-\$9,700) and 40% of women reported having completed high school or less (39% had completed some college/vocational degree and 21% a college degree/more). Women changed residences an average of 3.04 (SD = 2.71) times in the prior four years and almost 52% reported ever having used a domestic violence shelter. At Time 1, 22.50% of women reported being involved with a partner (violent or non-violent). Finally, 43.33% of the sample reported having had more than one violent intimate partner in their lifetime.

Procedure

Following study approval from the University Institutional Review Board, women with children were initially recruited in 2006-2010 through referrals from agencies providing services for women who had experienced IPV and through postings in local businesses, school campuses and social service agencies in Southeast Michigan and Southern Ontario. Recruitment materials included flyers and pamphlets, which provided a toll-free number to contact study staff. Women were enrolled in the study if they met the criteria of having experienced at least one act of IPV in the prior two years and if they had a child in the target age range for the study (4-6 years). Of the 150 people that called to inquire about the study, 25 did not meet study inclusion criteria and five declined to participate due to time constraints. Using block randomization, participants were placed in either the treatment (receipt of MEP) or the control (no intervention) group.

After providing written informed consent, women participated in a structured clinical interview. Female research assistants and graduate students in clinical psychology and social work, trained in clinical interviewing techniques and research ethics, administered the interviews. All interviewers received training from a licensed psychologist. Interviews were held at the participating service agencies, the research facilities of the university, participants' homes or at local community businesses in accordance with participants' preferences and safety

concerns. During the interviews, participants were asked about IPV, current mental health, trauma history, and demographic information. Interviews lasted one to two hours and participants were compensated \$20 for their time. All rights of the participants were protected.

The MEP intervention consisted of groups of six to eight women who met twice per week for five consecutive weeks. Details about the MEP and its theoretical foundations can be found elsewhere (S. A. Graham-Bermann, 2011). Program group leaders were either therapists at local mental health clinics and advocacy agencies, or graduate students in clinical psychology and social work. All therapists participated in a six-hour workshop where they received training in implementing the program. Therapists followed a training manual and received weekly supervision to enhance program adherence. Immediately following each session, group leaders wrote detailed process notes that included a description of the extent to which the treatment manual was followed. These notes were shared with the supervisor and then discussed in the subsequent group supervision. Concerns regarding content fidelity were examined in these meetings, including the establishment of a plan to integrate any missed material into the next therapy session. Following the completion of the intervention program or the five-week waiting period, participants completed a second clinical interview (time 2). Another follow-up interview was administered six to nine months later (time 3). This study added an eight-year follow-up assessment (time 4).

Given the amount of time elapsed between time 3 and 4 and the transient nature of this high-risk population, a range of strategies were used to locate and contact the participants. The first attempt to contact participants relied on contact information initially provided by participants at the first three measurement occasions, including telephone numbers, emails, and home addresses. If women were unable to be reached using this information, subsequent

procedures were implemented. These strategies included using social media (Facebook, Google+, and Instagram), online searches, and people-finding search engines. Women were then contacted either on the social media platform or the information collected was used to make attempted contact via phone or through letters sent to identified addresses. Information included in the follow-up recruitment attempts was intentionally vague to protect the privacy and safety of the women. The Institutional Review Board approved all recruitment procedures and materials.

Upon contact with study staff, women were provided an explanation of the fourth study phase and their participation was requested. Sixty-eight women were located and agreed to the follow-up interview. Similar procedures to the first three time points were followed for the interview process. Women were given the option to be interviewed at their residence if they deemed it appropriate and safe, or provided options to meet at local public places such as libraries and local business. Three of the participants no longer lived in Michigan; study staff travelled to their new states of residence to complete in-person interviews. Women were compensated in the amount of \$75 in cash for their participation in this phase of the study.

Measures

IPV re-engagement. Women's re-engagement was measured at each measurement occasion using a single question: "How many violent partners have you had in your lifetime?" During exploratory analyses of the data, some statistical concerns emerged. Given that the item asks about lifetime re-engagement with violent partners across the three time points, the count of such partners should either remain the same or increase. However, at time 3, eight women reported fewer violent partners than at time 1, and at time 4, 13 women reported having fewer partners than they did either time 1 or time 3. To address this discrepancy in lifetime re-engagement, if women reported fewer violent partners at time 3 than time 1, their time 1 count

was carried forward to replace their original time 3 count. Likewise, if their time 4 count was lower than either their time 1 or time 3 count, the higher value of the two time points was carried forward to replace the originally reported time 4 count.

IPV victimization. Frequency of IPV victimization over the prior year was assessed using the Revised Conflict Tactics Scale (CTS-2; Straus, Hamby, Boney-McCoy, & Sugarman, 1996). The CTS-2 is a 78-item measure that assesses physical assault, sexual coercion, psychological aggression, injury, and negotiation within intimate relationships. Respondents indicated the frequency of IPV events on a seven-point Likert-type scale ranging from 0 (never) to 7 (20 times or more). Although the CTS-2 assesses both IPV victimization and perpetration, due to the aims of this study only the 39 victimization items were administered. A total IPV score was created by summing all victimization questions except for the six negotiation items. The physical assault, sexual coercion, and psychological aggression subscale scores were created by taking the mean total of the items corresponding to each subscale. Internal consistency reliability for the present study was (α) .92. Internal consistency reliability for the subscales was: .91 for physical assault; .87 for sexual coercion; and .84 for psychological aggression.

Posttraumatic stress symptoms. The Posttraumatic Diagnostic Scale (PDS; Foa, Cashman, Jaycox, & Perry, 1997) was used to assess PTS over the prior month, using a Likert-type scale from 0 (not at all or only one time) to 3 (5 or more times a week/almost always). Participants were asked to identify their worst experience of IPV and report symptoms in relation to that experience. Subscales representing the three DSM-IV symptom domains of avoidance, reexperiencing, and arousal were created in addition to a total traumatic stress sum score, with a possible range of 0 to 51. Internal consistency reliability for the present study was (α) .89.

Internal consistency reliability (α) for the subscales was .76 for avoidance; .81 for reexperiencing; and .75 for arousal.

Childhood sexual abuse (CSA). CSA was assessed using the childhood sexual abuse item of the PDS (Edna B. Foa et al., 1997). At the first measurement occasion, participants were asked if they experienced “sexual contact when you were younger than 18 with someone who was 5 or more years older than you.” Responses were dichotomously coded: 0 (denied) or 1 (endorsed).

Cumulative interpersonal trauma. Cumulative history of interpersonal trauma was evaluated using items from the PDS (Edna B. Foa et al., 1997), which include questions about nonsexual and sexual assault, presence in a military combat or a war zone, imprisonment, and torture (CSA was not included). At each measurement occasion, participants endorsed or denied experiencing each type of traumatic event. At baseline, women were asked about lifetime history, whereas at the subsequent time points the timeframe of the items was since the last interview. A total interpersonal trauma score was created from the baseline measurement summing the seven traumatic event items. The cumulative interpersonal trauma score at time 3 was calculated by summing the baseline interpersonal score with the added interpersonal trauma reported since baseline. The cumulative interpersonal trauma score at time 4 was calculated by summing the baseline interpersonal score, the added interpersonal trauma reported between baseline and time 3, and the added interpersonal trauma reported between time 3 and time 4. A time 4 cumulative interpersonal trauma score was not able to be calculated for participants not present at time 3.

Depressive symptoms. Depressive symptoms were evaluated using the Center for Epidemiologic Studies Depression (CES-D; Radloff, 1977) 20-item self-report scale. Women

reported the frequency of symptoms over the past week on a 4-point Likert-type scale ranging from 0 (less than one day per week) to 3 (most or all of the time). Subscales representing four symptom domains (negative affect, positive affect, somatic, and interpersonal) were generated in addition to a total score. The total score was created using all 20 items, with a possible range of 0 to 60. Positive items were reverse-scored so that higher scores indicated greater symptoms. Internal consistency reliability (α) was .92 for the total scale; .82 for depressed affect; .80 for positive affect; .75 for somatic symptoms; and .57 for interpersonal (which only has 2 items).

Treatment group. Women assigned by block randomization to receive the MEP were considered treatment, while those who were not were designated as control. The treatment group was dummy coded: “0” for control and “1” for treatment.

Demographic variables. Women reported on their age, ethno-racial identification, education, relationship status, past domestic violence shelter use, employment status (employed/unemployed), monthly income, and housing instability (number of times moved).

Data Analysis

Linear multilevel modeling (MLM) was used to examine women with children’s risk factors (trauma exposure, mental health, and socio-demographic indicators) for IPV re-engagement across eight years and three measurement occasions. Given the correlations between repeated measures in longitudinal data (Luke, 2004; J. D. Singer & Willett, 2003), linear multilevel modeling in Stata 14 was selected to examine women’s changes in re-engagement over time. MLM can account for observations that are correlated across individuals, thus avoiding the underestimation of standard errors that can produce a statistically significant result when the null hypothesis is actually true (Type I error; Raudenbush & Bryk, 2002). Model assumptions were met including that errors at the level of the person-event and at the level of the

person were normally distributed. As part of standard practice in multilevel modeling (e.g., Raudenbush & Bryk, 2002) we first estimated a model with no independent variables in order to obtain variance components. These variance components then allowed us to calculate the intraclass correlation coefficient (ICC), a measure of the degree to which variation in the dependent variable is explained by time invariant, person specific characteristics. Here, the ICC is a measure of the degree to which differences between participants explains the variation in re-engagement (Raudenbush & Bryk, 2002). The following model was estimated: $y_{it} = \beta_0 + \beta_1(\text{IPV experienced} - \text{time variant}) + \beta_2(\text{CSA at baseline} - \text{time invariant}) + \beta_3(\text{Cumulative interpersonal trauma} - \text{time variant}) + \beta_4(\text{Traumatic stress symptoms} - \text{time variant}) + \beta_5(\text{Depressive symptoms} - \text{time variant}) + \beta_6(\text{Employment status} - \text{time variant}) + \beta_7(\text{Income} - \text{time variant}) + \beta_8(\text{Housing instability} - \text{time variant}) + \beta_9(\text{Wave}) + \beta_{10}(\text{Treatment group} - \text{time invariant}) + \beta_{11}(\text{Wave} * \text{treatment group}) + u_{0i} + e_{it}$. Missing data were handled via listwise deletion: give that there were multiple rows of data for each participant, participants were included in the analyses as long as they had complete data for at least one time point (49 women were missing at time 3 only; 52 were missing at time 4 only; 33 women were missing at both time 3 and 4).

A power calculation was conducted to determine the sample size needed to detect a medium effect size correlation between individual independent variables and re-engagement. Significance levels of (α) 0.05 and power of 0.8 were used as assumptions for the power analysis, which was calculated based on the method of expected data correlations (Champely, 2018; Cohen, 1988). In this sample of 120 women, expected correlations were at least 0.3, suggesting that only 84 distinct participants were needed for the study to be sufficiently powered. However, due to the longitudinal nature of the data, on average study participants were assessed approximately 2.5 times (due to study attrition), a total of 300 observations. Power calculations

must also consider the design effects of these intercorrelated observations (Public Health Action Support Team, 2019), which was estimated at 1.75 (from these 300 repeated observations). The effective sample size is thus estimated at 171 observations, suggesting that the current study has sufficient power to proceed with the planned analyses.

Results

Means and standard deviations for all study variables at each measurement occasion are summarized in Table 3.1 by treatment group. There were no differences between the treatment and the waitlist control group on any of the study's variables of interest at baseline (See supplemental Table 3.4 for bivariate correlations for all variables). Women who were not present at wave 4 did not have significantly different amounts of re-engagement at time 1 ($t = .86, p = .39$) or at time 3 ($t = .87, p = .39$) compared to those who were present at time 4.

Multi-level Model of Re-Engagement

The intra-class correlation coefficient without any predictors in the model was .73, which means that 73% of the variation in re-engagement over time was explained by time-invariant differences between women. The inclusion of random intercepts for individual participants was a significantly better approach than utilizing regression analyses without accounting for clustering, $\chi^2(1) = 132.55, p < .001$. MLM revealed that neither trauma exposure (including frequency of all types of IPV victimization across time, CSA, and cumulative interpersonal trauma over time) nor mental health concerns (including total PTS and depressive symptoms over time) were significantly associated with re-engagement across the eight years (see Supplemental Table 3.5). This was also true for employment, monthly income, and housing instability over time.

Results indicated that time was significantly associated with greater re-engagement at time 3 ($\beta = 0.29, p = .037$) and at time 4 ($\beta = 0.83, p < .001$). Treatment group alone was not

significantly associated with re-engagement: at baseline, the treatment and control groups did not significantly differ in re-engagement. However, findings revealed that the interaction between time and treatment was significant at time 4 ($\beta = -.79$, $p = .001$), indicating differences in re-engagement between treatment and control groups at the eight-year follow-up. Women who received the treatment had significantly fewer violent partners at this measurement occasion than those that did not. The interaction was not significant at time 3. The main effects can only be interpreted as being directly applicable to the control group given the significant interaction term.

Post-hoc Analyses

A post-hoc longitudinal MLM was estimated to further examine the potential contributing influence of the domains of IPV victimization, PTS, and depressive symptoms on women's re-engagement while also including the sociodemographic factors of interest (employment, income, and housing instability). Three subtypes of IPV (physical, sexual, and psychological IPV), three components of PTS (avoidance, reexperiencing, and arousal), and four domains of depression (positive affect, depressed affect, somatic symptoms, and interpersonal symptoms) all over time were included in the model in addition to the predictors included in the primary model. Delineation of the contributing roles of these subtypes/domains to re-engagement risk has the potential to provide more nuanced information on mechanisms of risk that may be directly actionable for future treatment tailoring and development.

Table 3.1 provides descriptive statistics for all predictors for the post-hoc analysis for each treatment group as well as across the entire sample. There were no baseline significant differences between the treatment and the control group on the study's variables of interest. For this follow-up analysis, a MLM was a better fit than a linear model, $\chi^2(1) = 130.43$, $p < .001$. Results revealed mixed trauma exposure findings (see Table 3.2): lower frequency of sexual IPV

and higher psychological IPV were significantly associated with more re-engagement over time. However, frequency of physical IPV victimization over time, CSA, and cumulative interpersonal trauma over time were not significantly associated with re-engagement.

Evaluation of the components of mental health, revealed that for PTS, having fewer reexperiencing symptoms was associated with having a greater re-engagement across time. PTS avoidance and arousal were not significantly associated with women's re-engagement over time. Of the four components of depression, more positive affect and more somatic symptoms were associated with greater re-engagement over time. Sociodemographic characteristics were associated with re-engagement in the second model. Here, lower income and less housing instability were associated with greater re-engagement. Women's employment was not significantly related to the outcome variable over time. Time alone was significantly related to more re-engagement at time 3 and at time 4, indicating significant changes in re-engagement for the control group over time. However, treatment group alone was not significantly associated with re-engagement, suggesting no differences in re-engagement between treatment and control groups at baseline. The interaction between time and treatment group was significant at time 4, such that women who received the treatment had significantly fewer violent partners at this measurement occasion than those that did not. The interaction was not significant at time 3.

Discussion

The present study sought to identify mechanisms of women with children's risk for IPV re-engagement by examining contributors of risk of lifetime re-engagement across eight years. This research is novel as it uniquely operationalized the IPV re-engagement construct, followed participants over an eight-year period, and rigorously assessed IPV victimization and re-engagement, cumulative interpersonal trauma, PTS, depression, employment status, income, and

housing instability over three measurement occasions. This study examined how participation in an established intervention affected re-engagement, a previously unexplored outcome of intervention research. This is a novel longitudinal examination of women with children's risk for IPV re-engagement with findings lending mixed support for proposed hypotheses.

Findings partially supported the first hypothesis of the expected relationship between IPV over time and re-engagement, suggesting that it is not the total amount of IPV victimization that is associated with re-engagement risk, rather it is specific forms of this victimization. Findings from the first model revealed no relationship between the total amount of IPV over time and women's re-engagement. However, findings from the post-hoc model revealed that less sexual IPV victimization but more psychological IPV was related to a greater re-engagement, consistent with previous cross-sectional findings (Stein et al., 2019). In contrast, physical IPV was not significantly associated with re-engagement in this sample, which also aligns with prior findings (Alexander, 2009; Stein et al., 2019). Certain types of IPV may be better understood by women to qualify as violence, and thus be clearly identified as a problematic relational behavior that may act as a deterrent from engaging in a relationship with someone. For example, greater awareness of sexual violence has emerged into public consciousness in part due to increased sexual assault awareness efforts (Vladutiu et al., 2011), which may have helped to make sexual IPV more readily identifiable and understood as unacceptable to women in this study. In contrast, less attention has been given to psychological IPV, which may be more challenging to identify and was correlated with higher rates of re-engagement in this sample. Although continued research is needed to fully understand the nuances of past IPV victimization on re-engagement risk, present findings preliminarily suggest the urgency of increasing awareness of what constitutes psychological IPV into prevention and treatment programs to mitigate re-engagement.

Surprisingly, findings did not support the second hypothesis that women's reported exposure to childhood sexual violence would be related to re-engagement. Past empirical work has established a link between CSA and risk for IPV re-engagement (Alexander, 2009; Stein et al., 2019; Vatnar & Bjørkly, 2008). The design of this study may account for the difference in findings. This work uniquely examined the contribution of CSA to re-engagement using a longitudinal design across eight years and simultaneously considered "what happened to you" (e.g., CSA) and "how are you doing" (e.g., PTS, depression). Perhaps, CSA served as a proxy for "how are you doing" in previous research that did not concurrently and more comprehensively assess symptomatology beyond affect dysregulation (Alexander, 2009).

Support was not found for the hypothesis that more cumulative interpersonal trauma is associated with greater re-engagement. In fact, findings revealed no significant relationship between cumulative interpersonal trauma and re-engagement in either model. This finding contrasts with earlier research linking a history of trauma exposure to sexual violence revictimization (Brenner & Ben-Amitay, 2015; Decker & Littleton, 2018), IPV victimization (Whitfield et al., 2003) and IPV re-engagement (Alexander, 2009; Cole et al., 2008; Stein et al., 2019). However, considering that this study concurrently assessed trauma history and mental health, the findings suggest that many of the effects of lifetime trauma exposure and re-engagement risk may operate through their influence on mental health symptoms. Future research is needed to determine if these symptoms mediate the relationship between lifetime trauma exposure and re-engagement. Furthermore, the present study did not examine cumulative interpersonal trauma during specific sensitive developmental periods. As a substantial body of evidence has identified the importance of early childhood experiences for lifetime relational

health (Schore, 2001), future research should examine the importance of cumulative interpersonal trauma exposure in childhood on risk for IPV re-engagement.

Study findings regarding the contribution of PTS to re-engagement were mixed. Total amount of PTS was not significantly associated with re-engagement, adding to previous findings (Cole et al., 2008; Stein et al., 2019). However, greater amounts of reexperiencing were related to having less re-engagement in this study, although avoidance and arousal were not. Limited past work has identified reexperiencing as a risk factor for experiencing future IPV (Kuijpers et al., 2012), but to our knowledge, no studies to date have examined the role of PTS symptom domains on re-engagement. Work examining the role of PTS symptom domains in mediating risk for sexual violence revictimization suggests that higher levels of hyperarousal may be associated with increased risk for sexual revictimization (Decker & Littleton, 2018). It is possible that PTS symptom domains differentially contribute to risk for future IPV given the disparate types of symptomatology present in the PTS diagnostic category (Schauer & Elbert, 2010). Perhaps, in the context of IPV more broadly, the persistent exposure to memories of violent partners serves a protective function to mitigate the replication of past negative experiences with intimate partners. However, despite this protective status of reexperiencing symptoms, we recognize that increasing PTS is not an appropriate intervention target. Thus, future work is needed to examine the processes by which high levels of reexperiencing lead to less re-engagement. Further understanding of how PTS reexperiencing buffers against re-engagement may serve to inform targets of intervention that could serve to mitigate risk while not diminishing well-being in the ways likely experienced by those with PTS.

As with PTS, total depressive symptoms were not associated with IPV re-engagement in this sample. Examination of the relationship between depressive symptom domains and re-

engagement revealed that lower levels of positive affect and higher levels of somatic symptoms over time were related to greater re-engagement. However, the domains of depressed affect and interpersonal symptoms were not significantly associated with re-engagement across the eight years. Findings suggest that it is not the presence of depressed affect that confers risk over time, but rather the absence of positive affect. Furthermore, women's somatic experiences seem to be a risk factor for re-engagement. This is an intriguing finding and a potentially a place of opportunity given the existence of somatically-informed intervention modalities including Mindfulness-based Contemplative Practices (Kabat-Zinn, 2003), Sensorimotor Psychotherapy (Ogden & Fisher, 2015), and Trauma-Informed Stabilization (Fisher, 2017b) all of which address the mind-body connection and somatization. No past research has examined relations between depression symptom domains and re-engagement. Thus, although these study findings are novel, they require further inquiry for rigorous interpretation and contextualization.

Employment status, income, and housing instability were included as sociodemographic variables of interest. Findings suggested that employment status was not significantly associated with re-engagement across the eight years, which coincides with limited previous findings (Alexander, 2009). Lower income was associated with greater re-engagement, which aligns with past research showing an inverse relationship between income and IPV (Ahmadabadi et al., 2017; Davies et al., 2015), though few studies to date have examined the specific role of income in the amount of re-engagement risk (Ørke et al., 2018). Women living in economically disadvantaged conditions are more likely to have higher levels of stress, experience greater unemployment, and have an exacerbated sense of hopelessness (Capaldi et al., 2012; Davies et al., 2015), which may then lead to increased IPV re-engagement. Yet, continued research is needed to delineate the precise mechanisms of income-related risk for IPV re-engagement for

women. Further, less housing instability was associated with higher levels of re-engagement. No previous work has examined the relationship between housing instability and re-engagement, and thus it would be presumptuous to draw a conclusion from a single finding. Continued work is needed to rigorously examine the role of these factors in conferring risk for IPV re-engagement.

Finally, results supported the efficacy of the MEP for the long-term reduction of IPV re-engagement in women with children. At the eight-year follow-up, women who received the treatment had significantly fewer violent partners than those in the control group. However, this was not true at the six-month follow-up. These are encouraging findings for cost-effective group intervention utilizing an empowerment perspective. It is hopeful that these 10 one-hour psychotherapy group sessions may be sufficient to begin to address women's risk for re-engagement, although the reasons why it was effective are unclear.

Limitations

This study provides novel information on what contributes risk and protection to women's IPV re-engagement, but it is not without limitations. The analyses were influenced by issues of missing data due to participant attrition across the eight years of the study. Specifically, it is likely that re-engagement data at time 4 was not missing at random, particularly in the treatment group, as the mean for re-engagement for this group at time 4 is lower than time 3. It is possible, therefore, that the significant interaction between time 4 and the treatment group is attributable to the IPV re-engagement data not being missing at random. Future studies would benefit from additional contact with participants between data collection waves to limit attrition. Although this research examined the novel construct of women's re-engagement, the outcome was based on a single item question. Variation in responses on the item across waves contributed to increased statistical noise and greater difficulty determining the true nature of the observed

relationships. Future work needs to focus on the development of a comprehensive assessment tool with greater reliability for assessing re-engagement. The present study also did not include any empirical assessment of information about the partners or relationships including the length of the relationship, the amount and type of IPV during the relationship, any contextual factors, or any information on reasons for staying with or barriers to leaving a violent partner. Further research should utilize assessments to comprehensively capture the nature and contextual factors associated with IPV. Additionally, this study did not measure women's IPV perpetration, which further limits important contextual information for understanding risk for being in a relationship with IPV. Future work should include assessments of potential IPV by both partners.

Although this study examined the contributions of PTS and depressive symptoms to re-engagement risk over time, future studies should examine additional and related mental health concerns such as bipolar and borderline personality disorder symptoms and if these symptoms mediate the relationship between initial IPV victimization and re-engagement. CSA was examined in this study but future research should assess for additional types of childhood trauma (e.g., witnessing IPV, neglect, physical abuse) with attention to the timing of the events. Future studies might also compare the MEP to different types of treatment rather to a control group. Finally, women in this study had children; findings may not generalize to those without children.

Conclusions

This study provides a rich examination of women's risk factors for IPV re-engagement using prospective methodology with eight-year longitudinal data. Findings suggest that it is not so much "what happened" (i.e., experiences of abuse) that create risk for re-engagement rather "how you are doing" following trauma (i.e., specific traumatic stress and depressive symptoms). This is hopeful as there are many evidence-based interventions shown to be effective for treating

PTS and depression following trauma. Furthermore, this study found support for the effectiveness of the MEP at reducing re-engagement in women who received the treatment versus controls over the eight years. Continued work is needed to identify which aspects of the MEP intervention addressed re-engagement. Isolation of these change mechanisms could inform larger-scale, cost-effective intervention with broad public health implications. Interventions that target IPV risk could help individuals foster a greater sense of personal efficacy and autonomy in establishing healthy and rewarding intimate relationships.

Table 3.1. Study variables over the three measurement occasions.

Variable	Time 1: Baseline		Time 3: 6 Months		Time 4: 8 Years	
	Treatment m (SD) / %	Control m (SD) / %	Treatment m (SD) / %	Control m (SD) / %	Treatment m (SD) / %	Control m (SD) / %
Re-engagement	1.50 (0.76)	1.72 (1.14)	1.70 (0.81)	2.27 (1.39)	1.60 (0.85)	2.45 (1.48)
> 1 violent partner	43.10%	43.33%	51.32%	63.33%	45.71%	75.76%
Employment	40.68%	36.07%	50.00%	45.71%	65.71%	72.73%
Monthly Income	1,280 (1,097)	1,411 (1,601)	2,086 (2,051)	1,379 (1,180)	3,234 (3,286)	2,107 (1,676)
Housing instability	3.31 (2.44)	2.78 (2.94)	3.36 (2.87)	2.54 (2.37)	3.34 (2.79)	2.76 (2.53)
Total IPV	201.79 (151.42)	180.64 (125.98)	28.11 (48.01)	50.14 (65.48)	40.00 (71.79)	30.50 (44.23)
Physical IPV	4.96 (5.23)	4.45 (4.18)	0.20 (0.61)	0.70 (1.49)	0.43 (1.12)	0.37 (1.20)
Sexual IPV	3.47 (5.33)	3.87 (5.82)	0.14 (0.48)	0.38 (1.03)	1.37 (3.34)	0.17 (0.56)
Psych IPV	12.58 (7.26)	11.48 (6.03)	3.19 (5.47)	4.73 (5.99)	3.06 (5.58)	2.85 (3.27)
CSA	44.07%	49.18%	-	-	-	-
CIT	1.47 (1.22)	1.57 (1.28)	2.13 (2.43)	2.15 (2.31)	4.50 (4.83)	3.76 (3.25)
PTS sx	21.86 (12.61)	22.08 (11.17)	15.68 (11.28)	13.00 (10.30)	15.83 (13.44)	19.41 (13.08)
Avoidance	8.23 (5.90)	8.24 (4.80)	6.76 (5.67)	4.94 (4.81)	6.71 (6.20)	7.88 (5.91)
Reexperiencing	6.51 (4.15)	5.93 (3.97)	3.40 (3.70)	2.76 (3.00)	3.14 (3.56)	4.06 (4.26)
Arousal	7.12 (4.39)	7.92 (4.36)	5.26(4.13)	5.29 (4.18)	5.80 (4.92)	7.38 (5.11)
Depressive sx	25.66 (13.26)	25.75 (14.04)	18.56 (11.40)	16.97 (9.85)	18.80 (11.40)	20.16 (12.81)
Positive affect	4.86 (3.40)	4.93 (3.25)	3.36 (2.98)	4.11 (3.50)	3.09 (3.01)	4.28 (3.03)
Depressed Affect	6.71 (4.18)	6.80 (4.13)	4.24 (3.31)	3.63 (2.84)	4.57 (4.15)	4.72 (4.17)
Somatic sx	7.17 (4.00)	7.27 (4.05)	5.94 (3.46)	5.03 (3.31)	6.49 (3.53)	5.91 (3.87)
Interpersonal sx	2.32 (1.81)	2.17 (1.87)	1.72 (1.58)	1.09 (1.17)	1.49 (1.70)	1.75 (1.67)

Note: There were no significant difference between the treatment and waitlist control groups on any of the indicator variables at baseline; CSA = childhood sexual abuse; CIT=cumulative interpersonal trauma

Table 3.2. Post hoc analysis: Linear MLM model estimating re-engagement over eight years using indicator subscales

Fixed Effects	b	ß	SE	Z	P	95% CI
Physical IPV	-0.006	-0.022	0.016	-0.39	0.693	[-0.037, 0.024]
Sexual IPV	-0.022	-0.085	0.011	-1.99	0.046	[-0.043, 0.000]
Psych IPV	0.024	0.160	0.009	2.59	0.010	[0.006, 0.043]
CSA	0.280	0.125	0.187	1.49	0.136	[-0.088, 0.647]
CIT	0.019	0.044	0.021	0.92	0.357	[-0.022, 0.060]
Avoidance	0.018	0.088	0.011	1.53	0.126	[-0.005, 0.040]
Reexperiencing	-0.034	-0.125	0.014	-2.46	0.014	[-0.061, -0.007]
Arousal	0.001	0.004	0.017	0.06	0.953	[-0.031, 0.033]
Positive affect	-0.037	-0.109	0.018	-2.08	0.038	[-0.072, -0.002]
Depressed affect	-0.030	-0.109	0.019	-1.61	0.108	[-0.067, 0.007]
Somatic sx	0.047	0.161	0.017	2.75	0.006	[0.013, 0.080]
Interpersonal sx	0.033	0.050	0.033	0.98	0.330	[-0.033, 0.098]
Employment	-0.084	-0.038	0.099	-0.85	0.394	[-0.277, 0.109]
Income	-0.000	-0.106	0.000	-2.49	0.013	[0.000, 0.000]
Housing instability	-0.041	-0.098	0.019	-2.12	0.034	[-0.079, -0.003]
Wave 3	0.272	0.305	0.114	2.38	0.017	[0.048, 0.496]
Wave 4	0.816	0.914	0.135	6.04	0.000	[0.551, 1.081]
Treatment group	-0.242	-0.109	0.182	-1.33	0.184	[-0.598, 0.115]
Wave 3 * Treatment	-0.142	-0.203	0.135	-1.05	0.294	[-0.406, 0.123]
Wave 4 * Treatment	-0.522	-0.747	0.158	-3.31	0.001	[-0.831, -0.212]
Constant	1.698		0.215	7.90	0.000	[1.277, 2.119]
Random effects	Estimate		SE		95% CI	
Person level variance	0.798		0.119		[0.595, 1.069]	
Residual variance	0.113		0.017		[0.084, 0.152]	

*CSA=Childhood sexual abuse; CIT=Cumulative interpersonal trauma; SE=Standard Error; CI=Confidence Interval

Figure 3.1. Moms' Empowerment Program CONSORT flow diagram.

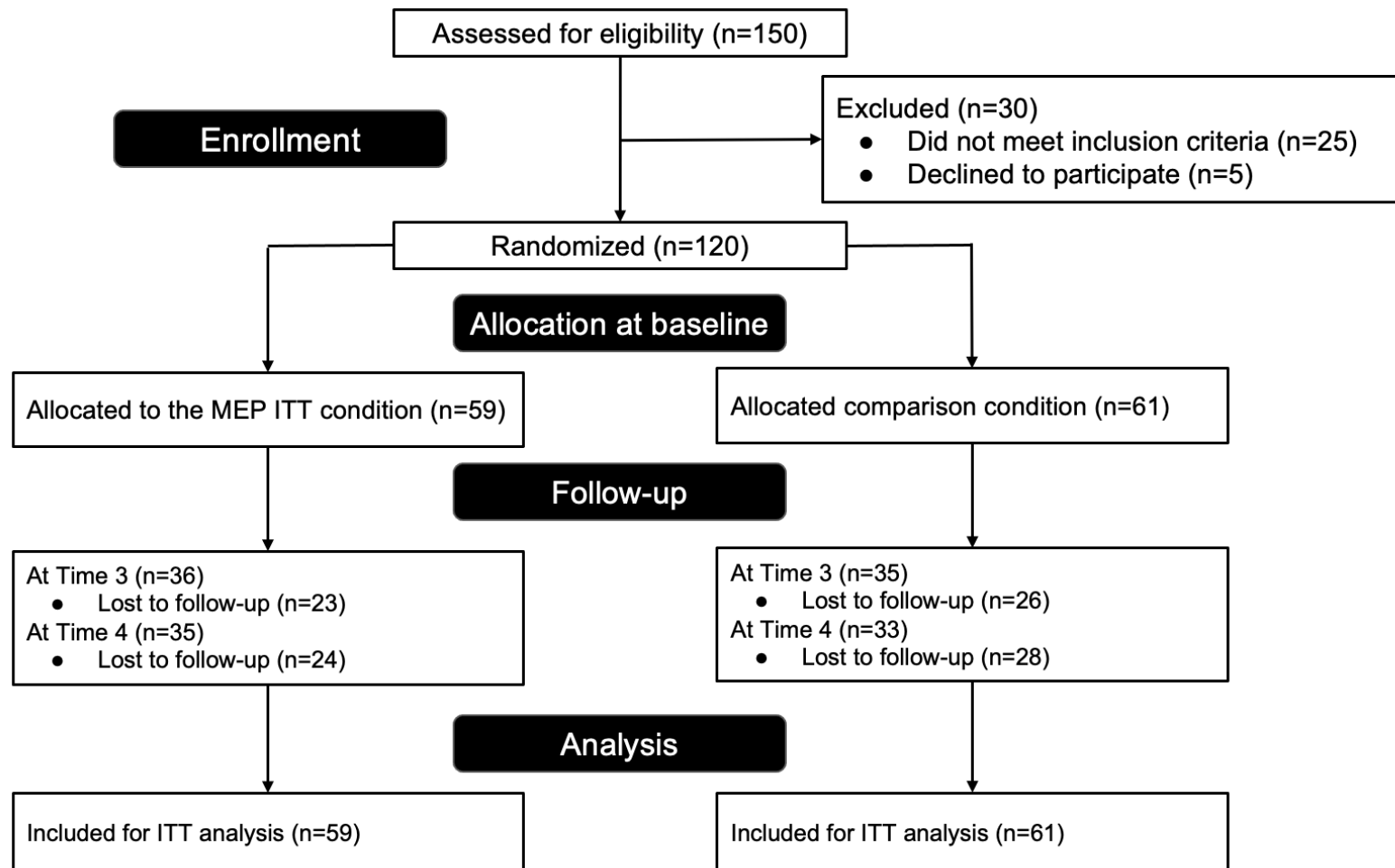


Table 3.3. Correlation matrix of primary study variables (Supplemental table)

Variable	1	2	3	4	5	6	7	8	9
1. Re-engagement	-								
2. IPV Victimization	.02	-							
3. CSA	.17**	.10	-						
4. CIT	.14*	-.01	.34***	-					
5. PTS	-.03	.42***	.29***	.25***	-				
6. Depression	.01	.38***	.17*	.16*	.71***	-			
7. Employment	-.20**	-.20**	-.23***	.05	-.16*	-.23***	-		
8. Income	-.16**	-.19**	-.12*	.23***	-.16*	-.12	.36***	-	
9. Housing instability	.01	.03	.27***	.19**	.20**	.22**	-.07	-.04	-
10. Treatment group	-.21**	.02	-.05	.03	-.02	-.00	-.02	.12	.12
11. Time	.17*	-.57***	-	.34***	-.21**	-.25***	.23***	.26***	-.00

*p<.05, ** p<.01, ***p<.001; CSA=Childhood sexual abuse; CIT=Cumulative interpersonal trauma;

Table 3.4. Correlation matrix of study variables used in post-hoc analysis (supplemental table)

Variable	1	2	3	4	5	6	7	8
1. Re-engagement	-							
2. Physical IPV	.01	-						
3. Sexual IPV	-.07	.54***	-					
4. Psychological IPV	.05	.72***	.49***	-				
5. CSA	.17	.11	.16*	.02	-			
6. CIT	.14*	-.01	.11	-.07	.34***	-		
7. Avoidance	.01	.30***	.30***	.30***	.36***	.31***	-	
8. Reexperiencing	-.15*	.33***	.26***	.39***	.10	.09	.58***	-
9. Arousal	.03	.28***	.35***	.34***	.23***	.22**	.72***	.57***
10. Positive affect	-.07	.20*	.16*	.20**	.11	.12	.44***	.28***
11. Depressed affect	.00	.30***	.24***	.40***	.17*	.10	.59***	.49***
12. Somatic sx	.04	.24***	.25***	.29***	.14*	.20**	.58***	.51***
13. Interpersonal sx	.37***	.19**	.09	.22**	.11	.10	.48***	.37***
14. Employment	-.06	-.18**	-.12	-.20**	-.23***	.05	-.16*	.06
15. Income	-.14*	-.21	-.10	-.14*	-.12	.23***	-.14*	.14*
16. Housing instability	.11	.07	-.04	-.01	.27***	.19**	.23***	.11
17. Treatment group	.02	.02	.00	.00	-.05	.03	.01	.02
18. Time	-.32***	-.50***	-.34***	-.58***	-	.34***	-.12	.32***

*p<.05, ** p<.01, ***p<.001*CSA=Childhood sexual abuse; CIT=Cumulative interpersonal trauma;

Variable	9	10	11	12	13	14	15	16
10. Positive affect	.42***	-						
11. Depressed affect	.57***	.58***	-					
12. Somatic sx	.62***	.46***	.75***	-				
13. Interpersonal sx	.42***	.48***	.70***	.58***	-			
14. Employment	-.17*	-.26***	-.20**	-.15*	-.14*	-		
15. Income	-.14*	-.10	-.13*	-.08	-.01	.35***	-	
16. Housing instability	.17*	.14	.20**	.20**	.19**	-.07	-.04	-
17. Treatment group	-.09	-.09	.01	.05	.05	.02	.12	.12
18. Time	-.14*	-.18**	-.27***	-.15*	-.19**	.23***	.26***	-.00

*p<.05, ** p<.01, ***p<.001

Table 3.5. Linear multilevel model estimating re-engagement over eight years (supplemental table)

Fixed Effects	b	ß	SE	Z	P	95% CI
IPV Victimization	0.000	0.017	0.000	0.34	0.733	[-0.001, 0.001]
CSA	0.253	0.113	0.189	1.34	0.180	[-0.117, 0.623]
CIT	0.026	0.060	0.022	1.19	0.234	[-0.017, 0.068]
PTS	0.001	0.014	0.006	0.24	0.814	[-0.010, 0.012]
Depression	0.000	0.003	0.005	0.06	0.950	[-0.009, 0.010]
Employment	-0.125	-0.056	0.103	-1.21	0.226	[-0.328, 0.078]
Income	0.000	-0.041	0.000	-1.03	0.303	[0.000, 0.000]
Housing instability	-0.035	-0.084	0.020	-1.72	0.085	[-0.075, 0.005]
Wave 3	0.254	0.285	0.122	2.08	0.037	[0.015, 0.493]
Wave 4	0.740	0.830	0.142	5.20	0.000	[0.461, 1.020]
Treatment group	-0.231	-0.104	0.186	-1.24	0.213	[-0.595, 0.133]
Wave 3 * Treatment	-0.109	-0.156	0.142	-0.76	0.445	[-0.388, 0.170]
Wave 4 * Treatment	-0.552	-0.791	0.165	-3.34	0.001	[-0.876, -0.229]
Constant	1.729	-	0.211	8.20	0.000	[1.316, 2.142]
Random effects	Estimate		SE		95% CI	
Person level variance	0.829		0.124		[0.619, 1.111]	
Residual variance	0.135		0.020		[0.101, 0.181]	

*CSA=Childhood sexual abuse; CIT=Cumulative interpersonal trauma; SE=Standard Error; CI=Confidence Interval

Chapter IV

Predictors of Experiencing Intimate Partner Violence Victimization in a Community

Sample of Emerging Adults

Intimate partner violence (IPV) victimization is a widespread social problem that includes the acts or threats of physical, sexual, and emotional violence by an intimate partner, including stalking, psychological aggression, and coercive behavior tactics (Black et al., 2011). Findings from the Center for Disease Control's 2015 National Intimate Partner and Sexual Violence Survey (S. G. Smith et al., 2018) suggest that approximately 36% of women and 34% of men will experience IPV in their lifetime (including physical violence, sexual violence, and/or stalking). Emerging adulthood (ages 18 to 25) is a unique developmental period (Arnett, 2000) when IPV involvement may peak or solidify (Rennison & Rand, 2003; White et al., 2009) as this is a time when intimate relationships become more serious (Arnett, 2000) and substance use also peaks (Jackson et al., 2008; White et al., 2009). IPV involvement during this period is associated with catastrophic social and personal costs that may extend throughout the lifetime (Liebschutz & Rothman, 2012). Yet, little is understood about what confers risk for experiencing IPV victimization versus no IPV involvement in emerging adulthood, especially outside of samples of college students (Coker et al., 2016). This study examines a community sample of 645 emerging adults (EAs) presenting at the emergency department (ED) to identify the trauma exposure and mental health mechanisms of risk for experiencing IPV victimization, while controlling for known sociodemographic variables.

The theory of syndemics (Singer et al., 2017) provides a “disease” to “disease” and social context to “disease” interaction framework that can help to delineate the risk mechanisms for experiencing IPV victimization. Syndemics suggests that diseases or social problems overlap and synergistically contribute to each another, resulting in both the clustering of and interaction between the problems (Singer, 2009; Singer, Bulled, Ostrach, & Mendenhall, 2017; Singer & Clair, 2003). The theory further specifies that social environments in which these problems occur – namely structural inequality and discrimination - contribute to and exacerbate the interaction of these problems (Singer, 2009; Singer, Bulled, Ostrach, & Mendenhall, 2017). Robust evidence shows that IPV victimization, trauma exposure (e.g. non-partner violence), and mental health concerns (e.g. anxiety, IPV aggression, alcohol use, and marijuana use) are co-occurring social problems, and that social contextual factors (e.g. sex, household income, and race) affect all three social problems and severity outcomes (Golden et al., 2013; James et al., 2013; Lipsky et al., 2009), suggesting synergistic relationship between these three social problems. The present study will examine the co-occurrence of trauma exposure and mental health, while controlling for sociodemographic factors as potential mechanisms of risk for IPV victimization. In addition, this work will explore the interaction between distinct but co-occurring mental health concerns (alcohol use and IPV aggression and alcohol and marijuana use) as well as the interaction between mental health and trauma exposure as a potential syndemic relationship increasing odds for experiencing IPV victimization compared to no IPV involvement in emerging adulthood.

Trauma exposure

Non-partner violence (NPV) victimization. NPV is a type of trauma exposure identified as a potential risk factor for IPV victimization (Murphy, 2011; Rodriguez-Menés et al., 2014; Taylor et al., 2008). A study using the Finnish National Crime Victim Survey (N = 25,927)

found that individuals with IPV victimization were significantly more likely to have non-IPV violent victimization than those without IPV victimization (Tanskanen & Kivivuori, 2021). In other work using data from the National Longitudinal Study of Adolescent to Adult Health (N = 11,928; 18–34 years of age), experience of youth violence victimization was associated with increased with of IPV victimization in first relationships, independent of the developmental timing of the relationship (Kuhl et al., 2015). Community violence victimization, a more all-inclusive category of NPV, has also been robustly linked with increased odds of experiencing IPV victimization (Murphy, 2011; Taylor et al., 2008). Continued work is needed to delineate the relationship between NPV victimization and IPV victimization in early adulthood, and the potential syndemic relationship between NPV and known mental health concerns on IPV victimization risk.

Mental health

Anxiety. Anxiety is a prevalent mental health concern in EAs (Gros et al., 2010) though limited work has examined the role of anxiety in IPV victimization risk (Golding, 1999; Gros et al., 2010; Kuijpers et al., 2011). Using the Fragile Family and Child Wellbeing data, Suglia, Duarte, and Sandel (2011) found that having experienced IPV victimization in the prior 12 months was associated with 2.92 times the odds of having generalized anxiety disorder compared to those who had not. A systematic review and meta-analysis (Trevillion et al., 2012) found that the pooled odds ratio for lifetime IPV victimization was 4.08 for among women with anxiety disorders as compared to those without. Further, in a more recent population-based study in the UK (Chandan et al., 2019), women who had experienced IPV victimization had an 1.99 adjusted incidence rate ratio of anxiety increase compared to those without. Unfortunately, to date, no work has examined, the potential predictive role of anxiety in IPV victimization risk. In a

systematic review of the prospective evidence for revictimization by IPV, Kuijpers and colleagues (2011) were unable to identify any studies that examined the potential role of anxiety in IPV victimization risk. Empirical research on anxiety as a potential risk factor for IPV victimization in EAs is needed.

IPV aggression. The majority of EA involvement in IPV has been found to be bidirectional (i.e. both IPV victimization and aggression; Cunradi et al., 2020; Renner & Whitney, 2012) rather than unidirectional (only victimization or aggression), suggesting that IPV aggression may be a key risk factor for IPV victimization (Baker & Stith, 2008; Renner & Whitney, 2012). The recent theory of the typologies of intimate partner violence (Johnson, 1995, 2008), suggests that the most common type of IPV is when a conflict gets “out of hand” and is the result of interpersonal dynamics of conflict management (known as "situational couples violence"; Johnson, 2008). Rather than a relationship being characterized by terroristic control of a partner, this IPV consists of isolated episodes of conflict escalation that result in the use of violence (known as “intimate partner terrorism”). From this standpoint, IPV aggression may be partially therefore attributable to difficulties with emotion regulation and coping (Bliton et al., 2016; Shorey et al., 2011) and may function as a related stress response in an attempt to mitigate the internal discomfort (Endler, 1997; Ngo et al., 2018) and thus is a mental health concern. Continued work is needed to understand the role of IPV aggression in odds of experiencing IPV victimization in emerging adulthood, especially as it overlaps with other mental health concerns such as alcohol use.

Alcohol use. Alcohol use peaks in emerging adulthood (Jackson et al., 2008; White et al., 2009) and evidence suggests an association with IPV victimization, though the direction of this relationship remains not well understood (Dardis et al., 2021; Devries et al., 2014; Kuijpers et

al., 2011; Shorey et al., 2011; Testa et al., 2004). Empirical findings consistently show a high co-occurrence of IPV victimization and alcohol use, including that high levels of IPV among substance use clinical samples of women (Chermack et al., 2000) and high levels of alcohol use among individuals residing in domestic violence shelters (Kaysen et al., 2008; Martin et al., 2008). For example, in a systematic review, an estimated 22-72% of women in shelters were found to have a current or past problem with alcohol (Schumacher & Holt, 2012). Furthermore, results of a nationally representative sample of 1,052 showed that mothers with IPV victimization have higher rates of alcohol use than those with no IPV (Ouellet-Morin et al., 2015). Yet, studies examining the role of alcohol in creating risk for IPV victimization have been mixed (Dardis et al., 2021; Devries et al., 2014; Kuijpers et al., 2011; Shorey et al., 2011; Testa et al., 2004). For example, cross sectional evidence suggests a strong link between alcohol use and IPV victimization (Devries et al., 2014). Vatnar and Bjørkly (2008) found that women's substance use was related to physical and psychological victimization, but not to sexual victimization. In another study, Fowler (2007) showed that women's number of years of alcohol use was significantly related to experiencing IPV physical abuse victimization. In contrast, other work revealed that substance use was only associated with IPV victimization when the sample was analyzed by racial groups (Nowotny & Graves, 2013). Specifically, support was found for alcohol as a predictor of IPV victimization in Black and Latina women, but not for White counterparts, suggesting the importance of controlling for race when examining the role of alcohol use on IPV victimization risk. However, other work has not found support for any association between alcohol use and IPV victimization (Dardis et al., 2021; Gilbert et al., 2012; Lipsky, Caetano, Field, & Larkin, 2005; Testa, 2004). In a longitudinal study of a representative sample of low-income women in an emergency department, binge drinking at baseline did not

predict any type of subsequent IPV victimization at 6- or 12-month follow-up (Gilbert et al., 2012). Likewise, Renner and Whitney (2012) found no significant relationship between alcohol use and IPV victimization using a sample of young adults from the National Longitudinal Study of Adolescent Health. Continued work is needed to discern the relationship between alcohol use and IPV victimization risk especially in emerging adulthood when alcohol use peaks (Jackson et al., 2008; White et al., 2009).

Marijuana use. Limited work has examined the relationship between marijuana use and IPV victimization (Shorey et al., 2016), despite marijuana being one of the most used substances in EAs (Johnston et al., 2013) and the most widely used drug among alcohol users (Substance Abuse and Mental Health Service Administration, 2014). Findings in adolescence are mixed: marijuana use was not associated with IPV victimization risk in a sample of 9,421 adolescents from National Longitudinal Study of Adolescent Health (Reingle et al., 2012). In contrast, a study of 27,758 high school students revealed that both alcohol and marijuana use were each associated with higher odds of IPV victimization (Parker et al., 2016), though an interaction between alcohol and marijuana use was not explored. The limited work examining the role of marijuana use in IPV victimization in adults is also very mixed. In a sample of college aged women, marijuana use was associated with increased odds of psychological and sexual IPV victimization (Shorey et al., 2016). However, in another study of adult women, marijuana use was associated with 4.3 decreased odds of experiencing sexual IPV coercion victimization in another study, but was not associated with physical violence victimization (Stuart et al., 2013). To date, no research has examined the role of marijuana use on IPV victimization risk in a community sample of EAs comprised of both men and women. Given the prevalence of

marijuana use and IPV victimization in this population, urgent attention is needed to discern the nature of this relationship in emerging adulthood.

Co-occurring mental health risk: potential synergistic impact on IPV victimization risk

Despite the above-cited high rates of alcohol use, marijuana use, and IPV aggression in emerging adulthood, little attention has been given to examining the potential interactive effects of these mental health concerns on odds for experiencing IPV victimization. In fact, none of cited literature examines either the interaction between alcohol and marijuana use or alcohol and IPV aggression on odds of experiencing IPV victimization despite alcohol's known role for decreasing inhibitions and influencing the interpretations of environmental and relational cues (Epstein-Ngo et al., 2013, 2014; Steele & Josephs, 1988). Furthermore, co-occurring mental health risks such as alcohol and marijuana use may exacerbate one another (Yurasek et al., 2017), potentially creating a synergistic impact on risk for IPV victimization. Given the high rates of overlap between alcohol use, marijuana use, and IPV aggression, research is needed that accounts for these as co-occurring and overlapping phenomena.

Study aims and hypotheses

This study examines the factors of trauma exposure and mental health that may contribute risk for experiencing IPV victimization in a community sample of EAs while controlling for known sociodemographic contributors of risk (sex, household income, and race; Capaldi et al., 2012; Cummings et al., 2013; Golden et al., 2013; Lipsky et al., 2009; Renner & Whitney, 2012; Ryan C. Shorey et al., 2008; Williams et al., 2016). The aim of this work is to delineate the independent and cumulative roles of trauma exposure (NPV victimization) and mental health concerns (anxiety, IPV aggression, alcohol use, and marijuana use) on odds of

experiencing IPV victimization versus no IPV victimization in a cross-sectional sample of EAs.

Specific hypotheses are as follows:

Hypothesis 1. Trauma exposure (NPV victimization) will be associated with greater odds for experiencing IPV victimization rather than no IPV victimization.

Hypothesis 2. The mental health concerns of IPV aggression and alcohol use will each be associated with greater odds for experiencing IPV victimization.

Research question 1: Will the mental health concerns of anxiety and marijuana use be associated with greater odds of experiencing IPV victimization?

Hypothesis 3. There will be a syndemic relationship between trauma exposure (NPV victimization) and mental health (anxiety, IPV aggression, alcohol use, and marijuana use) such that those with NPV victimization and higher levels of mental health concerns, will have greater odds of experiencing IPV victimization than those without trauma exposure and lower levels of the same mental health concerns.

Research Question 2. Exploratory analyses will examine interactions between alcohol use and IPV aggression and alcohol use and marijuana use on odds of IPV victimization given the high rates of co-occurrence of IPV aggression and marijuana use with alcohol use in EAs.

Methods

Participants.

Participants (N = 645) were EAs (ages 18 - 25 years) who sought services for any reason at a Level 1 urban emergency department (ED) in Flint, Michigan. The mean age of the sample was 21.5 (SD = 2.3) and approximately 37% were women. Approximately 64% of participants identified as a person of color, whereas 36% as white. Education level varied: 28% had less than

a high school education, 40% completed high school, and 32% had attended some college or more. The majority of the EAs (67.30%) were not accessing the ED for injury related concerns, and of those who were, only 16.75% reported that the injury was caused by a fight, conflict, argument, or personal attack. Refusal rates did not differ by age but did by sex: men refusing at higher rates than women (24.7% vs. 18.0%, respectively, $\chi^2 = 7.93$, $p < .01$). In addition, those that identified as Black had a lower refusal rate (16.0%) than those that identified as White (32.3%; $\chi^2 = 49.30$, $p < .001$).

Procedures

Study 3 utilizes baseline data collected from the screening phase of an intervention development project to reduce physical aggression and alcohol use. Participants were recruited from the Hurley Medical Center (HMC) in Flint, Michigan, a Level 1 urban ED. Participants who met the age criteria (18 - 25) and who were triaged within 30 minutes before the beginning or 30 minutes or more before the end of the research assistant's (RA) data collection shift in the ED were eligible to be approached.

Patients who presented with acute suicidal ideation or self-harm behaviors, acute sexual assault, acute psychosis, or a schizophrenia diagnosis, or those who were involved in a child abuse situation or in police custody were not eligible to participate. Additionally, patients who were unable to speak English, deaf, blind, illiterate or unable to speak, combative, legally married, too sick to approach, or unable to provide informed consent (e.g. unconscious, insufficient cognitive orientation, or developmental disability) were excluded from participation. Individuals who were on staff at Hurley, had previously refused participation two times, were already screened previously or currently enrolled in the study, or were participating in another study in Hurley ED were also ineligible. Finally, participants who were acutely intoxicated were

only approached after they were deemed clinically clear to provide consent by hospital staff. If the participant could not be approached right away due to extent of illness/injury, they were checked on again at a later time. Eligible EAs being seen for breathing difficulties (and a high pulse), were examined to see if they looked well enough to participate; if so, they were approached. If the patients did not look well enough to participate, they were checked on again at a later time. The Mini-Mental Status Exam (MMSE) was administered to any participant that received medication, had a head injury, or in any case the RA suspected that the patient could not give informed consent; a score of 18 or greater was considered “passing”. If a patient told staff to come back later, they were checked on twice, then excluded and moved on. Patients who refused to participate in the study were asked if they would provide information on their self-identified sex and race as well as reasons for refusing to participate. The University of Michigan and HMC IRB approved the study.

After completing a written consent, participants completed a self-administered tablet-based screening survey that took approximately 30-40 minutes. Questions included standardized measures of IPV, NPV perpetration and victimization, substance use, anxiety and depressive symptoms, trait mindfulness, and demographic questions. Participants received a gift valued at \$1.00 (e.g., puzzle book, playing cards, travel mug, pens) for their time. Data collection ran from December 2013 to December 2019.

Measures

IPV victimization. IPV victimization was measured using a modified version of the Revised Conflict Tactics Scales (CTS-2; Straus et al., 1996). The measure was modified to examine IPV over the past six months with any intimate partner (casual sexual partner, girlfriend/boyfriend, fiancée, or husband/wife). Participants responded to the number of times

specific acts occurred over the past six months on a Likert scale ranging from 0 “Never” to 6 “20 or more times”. A sum variable was created of the total amount of violent victimization experienced. A categorical variable was created to identify IPV victimization membership group based on the CTS items: 0 was assigned to participants that reported no IPV victimization and 1 to those who reported IPV victimization.

IPV aggression. IPV aggression was also measured using a modified version of the CTS-2 (Straus et al., 1996) as described above. Sample items included, “I beat my partner up” and “I punched or hit my partner with something that could hurt”. A sum variable was created of the total amount of IPV aggression reported, with possible score ranging from 0 – 126.

Non-partner violence victimization. NPV with someone who is not an intimate partner over the prior six months was measured using two items adapted from the Revised Conflict Tactics Scales (CTS-2; Straus et al., 1996). The administered items to assess for NPV included: “How many days did someone twist your arm, throw something that could hurt, push, shove, grab, slap, slam you against the wall?” and “How many days did someone kick, punch, beat up, hit you with something that could hurt, or threaten you with a knife or gun?”. Participants responded on a seven-point Likert scale ranging from 0 “Never” to 6 “More than 20 times”. A binary variable was created for NPV victimization: 0 corresponded to “No NPV victimization” and 1 to “NPV victimization”.

Anxiety. Anxiety symptoms were measured using items from the Brief Symptom Inventory (BSI; Derogatis, 2001; Derogatis & Melisaratos, 1983; Recklitis et al., 2006). Participants received three questions from the both the General and Panic anxiety subscales. Example questions include, “nervousness or shakiness inside” (general) and “spells of terror or panic” (panic subscale). Participants ranked their level of distress for each anxiety item over the

last week on a four-point Likert scale ranging from 0 (“Not at all”) to 4 (“Extremely”). Item responses were summed to create a total anxiety symptom score ($\alpha = .88$) with higher values reflecting higher symptomatology.

Alcohol. Alcohol use over the prior six months was assessed using the Alcohol Use Disorders Identification Test–Consumption (AUDIT-C; Bush, Kivlahan, McDonnell, Fihn, & Bradley, 1998). Participants rated consumption on a five-point Likert scale ranging from 0 “Never” to 4 “Daily/Almost Daily”. A sum score for the three items to measure alcohol consumption was created ($\alpha = .76$) where higher scores reflect more risky drinking. Prior research has identified a score of 3 or higher for women and 4 for men as indicative of likely risky drinking (Bradley et al., 2007; Bush et al., 1998).

Marijuana use. Marijuana use was assessed with the following question: “During the past 6 months, from today back to [date of 6 months ago], how many days did you use marijuana?”. Responses ranged from 0 “Never” to 5 “Every day or almost every day.” A categorical marijuana use variable was created: responses of “Never” were coded 0 for “No” and responses “1-5” were coded as “1” for “Yes” for marijuana use.

Sociodemographic factors. Sociodemographic measures included sex, household income, and race (defined as person of color).

Analyses

Analyses included descriptive statistics, bivariate analyses (t tests and chi-square), and multivariate logistic regressions using Stata 17 (StataCorp, 2021b) to examine contributors of experiencing IPV victimization versus no IPV victimization in this sample of EAs. Syndemic predictors included trauma exposure (NPV victimization), mental health (anxiety, IPV aggression, alcohol use, and marijuana use), and sociodemographic factors (sex, household

income, and race). Separate logistic regressions with all predictors were conducted to determine: 1) if there is evidence of syndemic relationship between trauma exposure and mental health on odds of experiencing IPV victimization by testing the interaction between NPV victimization and each mental health indicator on IPV victimization risk (models 1a-d); 2) if alcohol use moderated the relationship between IPV perpetration and IPV victimization (model 2); and 3) if marijuana use moderated the relationship between alcohol use and IPV victimization (model 3).

Results

Descriptive statistics and bivariate analyses

Among the sample of emerging adults (N = 645), 57.98% reported IPV victimization. Descriptive statistics for all study variables can be found in Table 4.1. EAs with youth violence victimization had a higher probability of experiencing IPV victimization than those with no youth violence victimization ($\chi^2(1) = 35.88, p < .001$). Those with no IPV victimization reported significantly lower levels of IPV perpetration compared to those with IPV victimization ($t(2,643) = -10.56, p < .001$). This was also true for anxiety ($t(2,643) = -4.19, p < .001$) and alcohol consumption ($t(2,643) = -3.30, p = .001$). EAs that reported marijuana use in the prior six months had a higher probability of experiencing IPV victimization than those with no marijuana use ($\chi^2(1) = 10.97, p < .01$).

Logistic regressions models

Model 1a-d: Testing the syndemic relationship between trauma exposure and mental health on IPV victimization risk. Results from the logistic regressions revealed main effect of IPV aggression and sex in each of the four models (See Tables 4.2 - 4.5). EAs with greater IPV aggression had 5.07 - 5.18 higher odds ($p < .001$) of experiencing IPV victimization compared to those with no IPV victimization. Identification as female was associated with .60 -

.61 of the odds ($p < .01$) of victimization compared to those that identified as male. There were no main effects of NPV victimization, anxiety, alcohol use, marijuana use, household income, or race on IPV victimization risk in these models. Furthermore, there were no significant interactions between NPV victimization and each of the mental health indicators (anxiety, IPV aggression, alcohol use, and marijuana use) in the models, largely suggesting that there is no evidence of a syndemic relationship between NPV victimization and mental health in this sample.

Model 2: Predicting IPV victimization with an alcohol use by IPV perpetration interaction. Results from the logistic regression including an interaction term of alcohol use by IPV perpetration revealed a main effect of IPV perpetration, such that higher amounts of IPV perpetration were associated with 3.71 increased odds ($p < .001$) of experiencing IPV victimization compared to those with no IPV victimization (see Table 4.6). Identification as female was associated with .60 decreased odds ($p = .005$) of victimization compared to those that identified as male. There were no significant main effects of youth violence victimization, anxiety, alcohol consumption, marijuana use, household income, or identification as a person of color on the odds of IPV victimization compared to those without IPV victimization.

Results showed a significant interaction between alcohol use and IPV perpetration on the odds of IPV victimization ($OR = 1.33$, $p = .006$). Recent work on logistic regression suggests that the coefficient from an interaction term may not provide accurate information about changes in slope across group membership (Karaca-Mandic et al., 2012). Therefore, we estimated predicted probabilities of the dependent variable at various values of the independent variables in order to better understand the interaction effect (StataCorp, 2021a). There was no significant interaction effect of alcohol use and IPV perpetration on IPV victimization at no IPV

perpetration ($\chi^2(1) = .91, p = .34$). However, amount of alcohol consumption made a significant difference for IPV victimization risk at low levels of IPV perpetration such that individuals who reported one violent act ($\chi^2(1) = 22.38, p < .001$), two violent acts ($\chi^2(1) = 25.24, p < .001$), and three violent acts ($\chi^2(1) = 6.90, p = .009$) over the prior six months reported greater IPV victimization risk as alcohol consumption increased. However, this was no longer the case for those reporting four violent acts ($\chi^2(1) = 3.07, p = .080$) and beyond.

Model 3: Predicting IPV victimization with an alcohol by marijuana use interaction.

Results from the logistic regression including an interaction term of alcohol use by marijuana use revealed a main effect of IPV perpetration, such that higher amounts of IPV perpetration were associated with 5.17 increased odds ($p < .001$) of experiencing IPV victimization compared to those with no IPV victimization (see Table 4.7). Alcohol consumption was also associated with 1.24 increased odds ($p = .041$) of IPV victimization. Identification as female was associated with .60 decreased odds ($p = .005$) of victimization compared to those that identified as male. There were no significant main effects of youth violence victimization, anxiety, marijuana use, household income, or identification as a person of color on the odds of IPV victimization compared to those without IPV victimization.

Results showed a significant interaction between alcohol use and marijuana use on the odds of IPV victimization ($OR = .78, p = .051$). A simple slope analysis revealed significant effects of not using marijuana such that those that didn't use marijuana had significantly higher IPV victimization risk as alcohol use increased ($\chi^2(1) = 4.76, p = .029$). This was not true of those that did use marijuana: IPV victimization risk did not increase as alcohol use increased ($\chi^2(1) = 0.24, p = .624$).

Discussion

The aim of this work is to delineate the independent and cumulative roles of trauma exposure (NPV victimization) and mental health concerns (anxiety, IPV aggression, alcohol use, and marijuana use) on odds of experiencing IPV victimization versus no IPV victimization while controlling for known sociodemographic contributors of risk. This work utilizes a unique novel community sample of EAs, a time when IPV victimization and substance use peak (Rennison & Rand, 2003; White et al., 2009). Prior studies on IPV victimization in emerging adulthood rely on convenience samples of college-going populations (Coker et al., 2016). The present study utilizes a community sample in that it includes both college- and non-college enrolled participants, allowing for greater generalizability of findings. Study results lend mixed support for the proposed hypotheses: trauma exposure (NPV victimization) was not significantly associated with IPV victimization, nor were the mental health concerns of anxiety or marijuana use alone. However, IPV aggression and alcohol consumption were each associated with increased IPV victimization risk. Findings did not support the hypothesis of a syndemic relationship between trauma exposure and mental health. Rather, findings suggested a synergistic relationship between IPV aggression and alcohol use and alcohol use and marijuana use on IPV victimization risk. Finally, identification as female compared to male was associated with decreased odds of experiencing IPV victimization.

Trauma exposure, operationalized in this study as NPV victimization, was not significantly associated with IPV victimization. This result stands in contrast to previous research which has established NPV as a potential risk factor for IPV victimization (Kuhl et al., 2015; Murphy, 2011; Rodriguez-Menés et al., 2014; Taylor et al., 2008). However, this prior work used samples that spanned larger and different developmental periods and thus was not limited to solely emerging adulthood, which is a unique developmental period (Arnett, 2000)

characterized by the emergence and consolidation of significant risk behaviors including substance use and IPV involvement (Jackson et al., 2008; Rennison & Rand, 2003; White et al., 2009). Thus, it's possible that while NPV may contribute significant risk for IPV victimization during other developmental periods, especially adolescence, this may not be in the case in EAs. EAs may not be as dependent on specific social contexts and environments as might be the case in prior developmental periods (i.e. middle and high school). It may be that experiences of NPV victimization when EAs have more diversity of social contexts is less consequential for IPV victimization risk than when social contexts are more fixed earlier and later in life.

Results partially supported the hypothesis that mental health would be associated with IPV victimization risk in this sample of EAs as specific mental health concerns were associated with IPV victimization risk and others were not. Evidence revealed no significant main effects of either anxiety or marijuana on IPV victimization risk. Although some prior work has established a potential link between anxiety and IPV victimization (Chandan et al., 2019; Suglia et al., 2011; Trevillion et al., 2012) overall past research has been very limited (Golding, 1999; Gros et al., 2010; Kuijpers et al., 2011). Prior evidence is also narrow (Shorey et al., 2016) and mixed for the role of marijuana use in IPV victimization risk. Some research has established a link between marijuana use and certain types of IPV victimization (Parker et al., 2016), while others studies have found no such relationship (Reingle et al., 2012; R. C. Shorey et al., 2016) or in fact decreased odds of IPV victimization with use (Stuart et al., 2013). However, this body of findings focused on both adolescents and college-going samples and thus may not generalize to community samples of EAs. Although longitudinal work is needed to better delineate the relationship between these mental health concerns and IPV victimization, it may be that anxiety

and marijuana use alone are not significant risk factors for IPV victimization when considered in conjunction with other more prominent drivers of risk.

Findings revealed significant main effects of IPV aggression and alcohol consumption on IPV victimization, though the latter main effect was significant only when also examining the interaction between alcohol and marijuana use. In every model examined, IPV aggression was significantly associated with between 3.71-5.18 increased odds of experiencing IPV victimization in this sample. This result coincides with past work that IPV aggression may be a key risk factor for IPV victimization (Baker & Stith, 2008; Renner & Whitney, 2012). Perhaps IPV aggression is the strongest indicator of IPV victimization risk as it indicates overall involvement in IPV, which has been shown to be primarily bidirectional in EAs (Cunradi et al., 2020; Renner & Whitney, 2012). Furthermore, it may be that IPV aggression is acting as a proxy for difficulties with emotion regulation and/or coping (Bliton et al., 2016; Shorey et al., 2011), which may be associated with high levels of relational lability and thus higher IPV victimization risk. Longitudinal research is needed to identify how IPV aggression specifically confers risk for IPV victimization in this population. In contrast, evidence was not found for a significant main effect of alcohol consumption on IPV victimization risk except when also examining the interaction between alcohol and marijuana use. This coincides with past findings on the mixed evidence for the role of alcohol use on IPV victimization risk (Dardis et al., 2021; Devries et al., 2014; Kuijpers et al., 2011; Shorey et al., 2011; Testa et al., 2004) despite high levels of alcohol use amongst those with IPV victimization (Chermack et al., 2000; Kaysen et al., 2008; Sandra L Martin et al., 2008). Although challenging to obtain, prospective longitudinal research is needed to more accurately determine if alcohol use is a risk factor for or a consequence of IPV victimization. Furthermore, findings from the current study may suggest that alcohol use is most

important when considered with other risk factors including IPV aggression and marijuana use, as discussed below.

Household income and race were not significantly associated with IPV victimization in this sample, although participant sex was. Specifically, across all models, women had approximately .60 decreased odds of experiencing IPV victimization compared to male counterparts. This contradicts some prior conclusions suggesting women experience physical IPV victimization at slightly higher rates than men (M. J. Breiding et al., 2014), though this was across the lifetime not limited to solely emerging adulthood. Another study found that in adolescence and young adulthood, identification as female (as compared to male) was associated with IPV victimization (Halpern et al., 2009). However, other work revealed that EA's IPV involvement in is primarily bidirectional (Cunradi et al., 2020; Renner & Whitney, 2012; Whitaker et al., 2007), suggesting that rates of IPV victimization should be roughly equivalent for men and women. Current study findings may be partially attributable to unequal group sizes, and thus caution should be exercised when interpreting the findings. Furthermore, prior work has suggested that the standardized measure used to assess IPV victimization in this sample, the CTS, may result in underreporting and overreporting of certain types of IPV across sexes (Chapman & Gillespie, 2019; Straus, 2012). Continued work using varied assessment tools is needed to understand the role of sex in IPV victimization risk, specifically as we continue to expand sex beyond a binary category.

Study findings that household income and race were not significantly associated with IPV victimization, partially contradict previous work. Robust evidence suggests a strong relationship between lower socioeconomic status and increased risk for IPV (Cunradi et al., 2002; L. Davies et al., 2015; Field & Caetano, 2004; Jewkes, 2002; Reichel, 2017). Individuals living in

economically disadvantaged conditions are more likely to have higher levels of stress, experience greater unemployment, be more socially isolated and have an exacerbated a sense of hopelessness, which may then lead to increased rates of IPV (Cummings, Gonzalez-Guarda, & Sandoval, 2013; Martin, Tsui, Maitra, & Marinshaw, 1999). The relationship between race and IPV victimization is less clear, with some studies finding that people of color experience more IPV victimization than white counterparts (Azziz-Baumgartner et al., 2011; Caetano, Field, et al., 2005; Lipsky et al., 2009; Miller-Graff & Graham-Bermann, 2016), while others have found no such relationship (Amy E. Bonomi et al., 2009). Given that the current study relied on a high-risk, community sample with low mean income and with a majority of EAs reporting being people of color, it may be that there was insufficient variation in these measures to detect meaningful statistical effects. Continued research is needed using more diverse samples to fully delineate the role of income and race on IPV victimization risk.

Findings did not support the hypothesis of a syndemic relationship between trauma exposure (NPV victimization) and mental health (anxiety, IPV aggression, alcohol use, and marijuana use) on IPV victimization in this sample as all interactions between NPV and the mental health indicators revealed non-significant associations. This evidence would suggest that the co-occurring social problems of trauma exposure and mental health concerns do not overlap to exacerbate IPV victimization risk while also considering important sociodemographic factors. However, these results should only be considered as an initial examination of the potential syndemic relationship between trauma exposure and mental health on IPV victimization risk, and therefore should not be interpreted as a robust finding. The current study was a secondary data analysis and therefore decisions on how to operationalize trauma exposure and mental health were largely driven by data availability. Future work would benefit from collecting theoretically-

and empirically driven retrospective, longitudinal data to rigorously explore the relationship between trauma exposure and mental health on IPV victimization risk. For example, trauma exposure should include robust measures of different types of trauma exposure across distinct developmental periods. It may be that the type and timing of trauma exposure are two key factors in the way that trauma interacts with mental health concerns to exacerbate IPV victimization risk but were two elements that were unavailable for examination in the current study.

Results revealed two separate synergistic relationships between mental health indicators on IPV victimization risk. Findings showed a significant interaction between IPV aggression and alcohol use such that at low levels of IPV aggression, higher levels of alcohol use were associated with significantly increased IPV victimization risk. This was true for individuals who reported one, two, and three IPV aggression acts in the prior six months. However, this was not true for those who reported four or more acts: here, IPV victimization risk remained high across all levels of alcohol consumption. This finding suggests that alcohol use confers most risk for IPV victimization for individuals very low levels of IPV aggression. It may be that alcohol's known role for decreasing inhibitions and influencing the interpretations of environmental and relational cues (Epstein-Ngo et al., 2013, 2014; Steele & Josephs, 1988) matters most for those who rarely utilize IPV aggression. Those utilizing IPV aggression frequently may already be primed to experience danger in their intimate relationships and alcohol use may not further exacerbate this perception in meaningful ways for victimization. Although prospective work is needed to better delineate the role that alcohol use has in exacerbating IPV victimization risk at low levels of IPV aggression, clinical interventions should begin to consider the importance of alcohol use for IPV victimization risk at all levels of IPV aggression.

Finally, findings revealed a significant interaction between alcohol and marijuana use on IPV victimization risk, such that those that didn't use marijuana had significantly higher IPV victimization risk as alcohol use increased. However, this was not true of those that did use marijuana: IPV victimization risk did not increase as alcohol use increased. This finding stands in contrast to previous work which suggests that co-occurring alcohol and marijuana use may exacerbate one another (Yurasek et al., 2017). However, to date, no other work has examined the combined effects of alcohol and marijuana use on IPV victimization risk in EAs. Future work would benefit from using longitudinal data with more comprehensive substance use assessments that include nuanced information on the timing of both substance use and IPV victimization.

Study results of the synergistic relationship between alcohol use and IPV aggression and alcohol and marijuana use on IPV victimization further highlight the complex relationship between alcohol use and IPV victimization previously established in literature (Dardis et al., 2021; Devries et al., 2014; Kuijpers et al., 2011; Shorey et al., 2011; Testa et al., 2004). Current findings suggest that alcohol use alone is an insufficient predictor of IPV victimization risk but needs to be considered in tandem with other important predictors of victimization risk. Future work should continue to consider the synergistic relationships between alcohol and other indicators of risk for IPV victimization in addition to examining alcohol use as a potential mediator between other indicators of risk and IPV victimization.

Limitations

The present study provides novel information on syndemic factors that may contribute unique risk to experiencing IPV victimization compared no IPV victimization. However, despite the strengths of this study, there are some limitations. This research relied on cross-sectional data, which precludes the drawing of causal inferences. Future work should focus on prospective

longitudinal designs that would allow for the identification of potential causal mechanisms for IPV victimization risk. Furthermore, given that recruitment for the current study was done in the ED, the length of the survey administered to participants had to be limited to accommodate the demands of the hospital. Future research would benefit from longer interviews to facilitate the administration of more complete standardized measures, specifically for mental health concerns. In addition, the current study did not include a measure of posttraumatic stress, which would be vital to include in future research given its association with IPV involvement (Dutton et al., 2006; Ehrensaft, Moffitt, & Caspi, 2006; Golding, 1999). Finally, this research may have been limited by its conceptualization and operationalization of syndemic predictors. Although findings suggest that there is likely no syndemic relationship between trauma exposure and mental health on IPV victimization in this sample, it appears that there may be a syndemic relationship between IPV aggression and alcohol use on victimization. Future research would benefit from continuing to refine the potential syndemic factors that may contribute risk for IPV victimization.

Conclusion

This study examines the independent and cumulative roles of trauma exposure (NPV victimization) and mental health concerns (anxiety, IPV aggression, alcohol use, and marijuana use) on odds of experiencing IPV victimization versus no IPV victimization in a community sample of EAs recruited in an urban emergency department. Study findings revealed that IPV aggression was significantly associated with between 3.71-5.18 increased odds of experiencing IPV victimization. Identification as female compared to male was associated with approximately .60 decreased odds of experiencing IPV victimization. Evidence did not support a syndemic relationship between trauma exposure and mental health. Rather, results suggest a synergistic relationship between IPV aggression and alcohol use and alcohol use and marijuana use on IPV

victimization risk. At low levels of IPV aggression (1-3 acts in the prior six months), alcohol use increases IPV victimization risk, but this does not hold for four acts or more. Here, IPV victimization risk remained high across all levels of alcohol consumption. Results also showed that those that didn't use marijuana had significantly higher IPV victimization risk as alcohol use increased. However, this was not true of those that did use marijuana as IPV victimization risk did not increase as alcohol use increased. Current findings suggest that alcohol use is an important but insufficient predictor of IPV victimization risk alone but needs to be considered in tandem with other important indicators of risk.

Table 4.1.

Sample descriptive statistics by victimization group (N=645)

	No IPV	IPV	
	Victimization	Victimization	t / X²
	(n=271, 42.02%)	(n=374, 57.98%)	
	M(SD) / n(%)	M(SD) / n(%)	
Youth violence victimization			35.88***
No	258 (46.82%)	293 (53.18%)	
Yes	13 (13.83%)	81 (86.17%)	
IPV perpetration	.25 (.89)	7.35 (11.04)	-10.55***
Anxiety	2.74 (4.31)	4.36 (5.24)	-4.2***
Alcohol Consumption	1.60 (2.30)	2.26 (2.69)	-3.3**
Marijuana Use			10.97**
No	164 (48.09%)	177 (51.91%)	
Yes	107 (35.20%)	197 (64.80%)	
House Income	\$3,080 (4,270)	\$3,085 (4,284)	-0.01
Sex			3.14
Men	178 (39.73%)	270 (60.27%)	
Women	93 (47.21%)	104 (52.79%)	
Person of Color	153 (25.16%)	236 (38.82%)	3.41

p < .01, * p < .001

Table 4.2.

Model 1a: Logistic regression examining syndemic predictors of IPV victimization: NPV
victimization * anxiety

	Odds ratio	SE	z	P> z	[95% CI]	
NPV victimization	2.870606	1.902289	1.59	0.112	.7832505	10.52075
Anxiety	.990686	.0332796	-0.28	0.781	.9275601	1.058108
NPV * Anxiety	.9616995	.0953713	-0.39	0.694	.7918193	1.168026
Alcohol Consumption	1.04677	.0621065	0.77	0.441	.931855	1.175857
IPV aggression	5.186978	.8129812	10.50	0.000	3.815059	7.052247
Marijuana Use	1.173315	.3334033	0.56	0.574	.6722667	2.047801
Sex	.3911045	.1274388	-2.88	0.004	.2065069	.740715
House Income	1.000005	.0000309	0.16	0.874	.9999443	1.000066
Persons of Color	.9918173	.266459	-0.03	0.976	.5858011	1.679242

Table 4.3.

Model 1b: Logistic regression examining syndemic predictors of IPV victimization: NPV
victimization * IPV aggression interaction

	Odds ratio	SE	z	P> z	[95% CI]	
NPV victimization	2.105135	1.249239	1.25	0.210	.6578943	6.736026
IPV aggression	5.077974	.809649	10.19	0.000	3.715113	6.940789
NPV * IPV aggression	1.357861	.962742	0.43	0.666	.3383312	5.449652
Anxiety	.9857384	.0319462	-0.44	0.658	.9250721	1.050383
Alcohol consumption	1.047317	.0622722	0.78	0.437	.9321086	1.176764
Marijuana Use	1.181254	.3357632	0.59	0.558	.6766981	2.062013
Sex	.3897824	.1268739	-2.89	0.004	.2059475	.7377137
House Income	1.000006	.0000308	0.21	0.834	.9999462	1.000067
Persons of Color	1.000652	.2688726	0.00	0.998	.5909725	1.694333

Table 4.4.

Model 1c: Logistic regression examining syndemic predictors of IPV victimization: NPV

victimization * alcohol use interaction

	Odds ratio	SE	z	P> z	[95% CI]	
NPV Victimization	3.726122	2.496166	1.95	0.051	.9961557	13.86286
Alcohol Consumption	1.059624	.0640008	0.96	0.338	.9413247	1.19279
NPV victimization* Alcohol	.786383	.2002935	-0.94	0.345	.4773437	1.295499
Anxiety	.9868592	.0314751	-0.41	0.678	.9270578	1.050518
IPV aggression	5.17842	.8079265	10.54	0.000	3.814121	7.030723
Marijuana Use	1.193568	.3392935	0.62	0.534	.6837185	2.083611
Sex	.4002041	.1302215	-2.81	0.005	.2115003	.7572723
House Income	1.000006	.0000307	0.20	0.840	.999946	1.000066
Person of Color	.9854579	.265333	-0.05	0.957	.5813711	1.670409

Table 4.5.

Model 1d: Logistic regression examining syndemic predictors of IPV victimization: NPV
victimization * marijuana use

	Odds ratio	SE	z	P> z	[95% conf. interval]	
NPV Victimization	3.817477	3.183822	1.61	0.108	.7444992	19.57441
Marijuana use	1.246095	.3681605	0.74	0.456	.6983325	2.223513
NPV * Marijuana use	.4875248	.5093548	-0.69	0.492	.0629044	3.778441
IPV aggression	5.157882	.8036601	10.53	0.000	3.800528	7.000013
Anxiety	.9856029	.0316255	-0.45	0.651	.9255269	1.049578
Alcohol consumption	1.04767	.062123	0.79	0.432	.9327198	1.176786
Sex	.3997501	.130338	-2.81	0.005	.2109869	.7573936
House Income	1.000007	.0000308	0.24	0.809	.9999471	1.000068
Person of Color	.9678893	.2631781	-0.12	0.904	.5680363	1.649207

Table 4.6.

Model 2: Logistic regression predicting IPV victimization with an alcohol use * IPV aggression interaction

	Odds ratio	SE	z	P> z	[95% CI]	
NPV victimization	2.520259	1.267097	1.84	0.066	.9407862	6.751485
IPV aggression	3.713154	.6436965	7.57	0.000	2.643518	5.215592
Anxiety	.9924745	.0325473	-0.23	0.818	.9306899	1.058361
Alcohol Consumption	.9325629	.0749809	-0.87	0.385	.7965975	1.091735
Alcohol * IPV aggression	1.336248	.1415876	2.74	0.006	1.085662	1.644673
Marijuana Use	1.131826	.3300608	0.42	0.671	.6390786	2.004497
Sex	.3971682	.1297694	-2.83	0.005	.2093417	.7535173
House Income	1.000008	.0000311	0.27	0.791	.9999474	1.000069
Persons of Color	.9083091	.2493635	-0.35	0.726	.5303325	1.555676

Figure 4.1.

Interaction between IPV aggression and alcohol use on IPV victimization probability

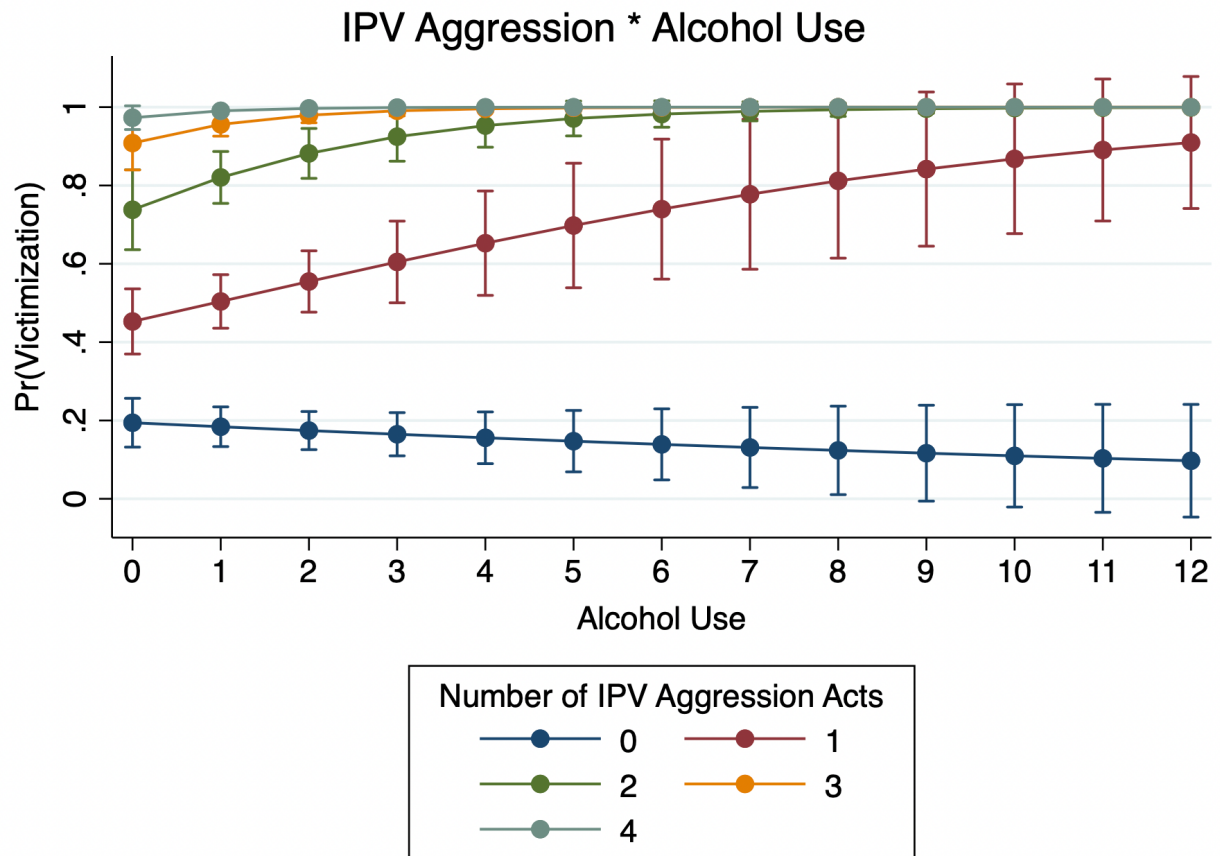


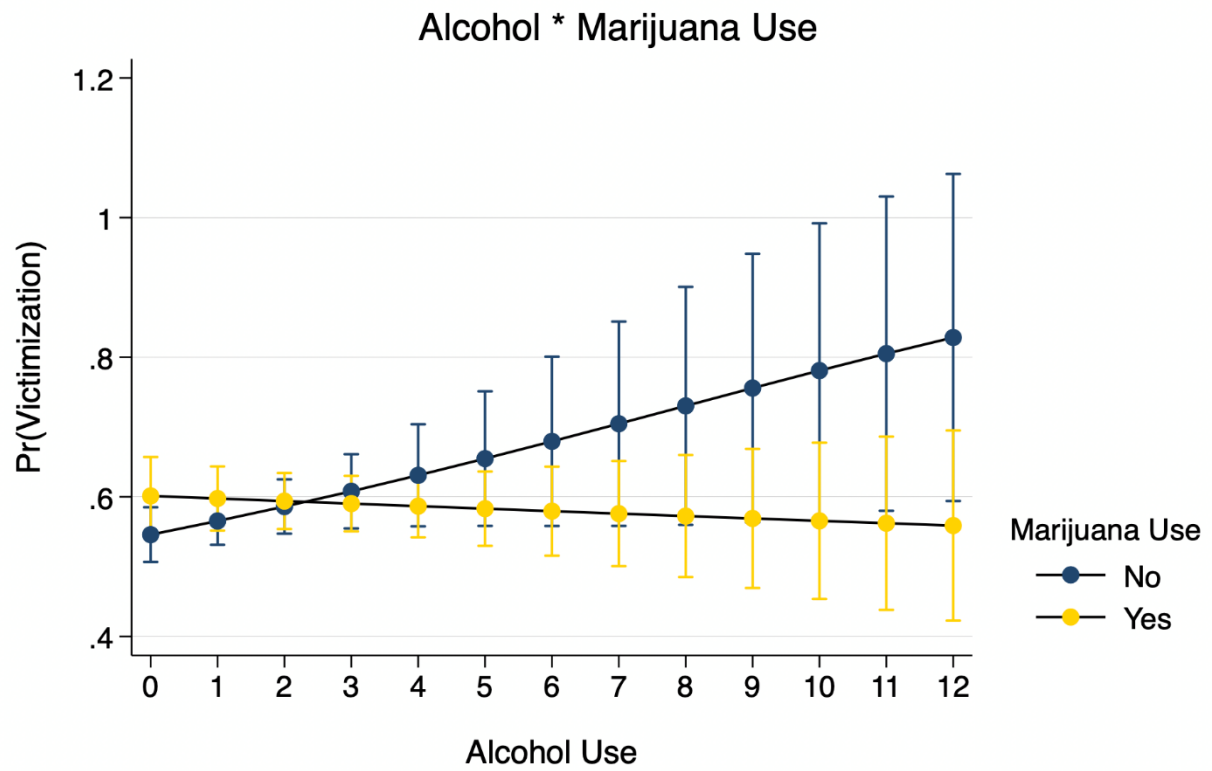
Table 4.7.

Model 3: Logistic regression predicting IPV victimization with an alcohol use * marijuana use interaction

	Odds ratio	SE	z	P> z	[95% CI]	
NPV Victimization	2.482062	1.226749	1.84	0.066	.9421273	6.539065
IPV aggression	5.169963	.8151801	10.42	0.000	3.795537	7.042092
Anxiety	.9900054	.0322117	-0.31	0.758	.9288426	1.055196
Alcohol Consumption	1.235398	.1275488	2.05	0.041	1.009078	1.512479
Marijuana Use	1.771096	.6243194	1.62	0.105	.8875431	3.534227
Alcohol * Marijuana Use	.7809738	.0987356	-1.96	0.051	.609568	1.000577
Sex	.4026763	.1319149	-2.78	0.005	.211888	.7652545
House Income	1.000005	.000031	0.16	0.875	.9999441	1.000066
Person of Color	1.01066	.2734977	0.04	0.969	.5946462	1.717715

Figure 4.2.

Interaction between alcohol and marijuana use on IPV victimization probability



Chapter V

Conclusion

Approximately 24 percent of women and 14 percent of men will experience IPV in their lifetime (Breiding, Chen, & Black, 2014), which includes the acts or threats of physical, sexual, and emotional violence by a current or former intimate partner, encompassing stalking, psychological aggression, and coercive behavioral tactics (Black et al., 2011). IPV is a pervasive social issue with broad individual and social implications (National Center for Injury Prevention and Control, 2003). Furthermore, chronicity of IPV across multiple partners is associated with more profound consequences than acute exposure (Beydoun et al., 2012). Despite the enormity of the individual and social costs of IPV, current treatments to reduce IPV victimization have been largely ineffective (Eckhardt et al., 2013) suggesting that the mechanisms of risk for this victimization remain mostly unknown. In particular, even though past trauma exposure, mental health concerns, and sociodemographic factors have been loosely associated with IPV victimization risk, prior empirical work has largely neglected to consider these factors concurrently and in a manner that would allow for the identification of actionable targets of intervention (e.g. symptoms that exacerbate risk versus diagnostic categories that are inherently heterogeneous in presentation). Research is needed to comprehensively delineate precise and actionable mechanisms of risk for IPV victimization to inform novel intervention targets and improve the efficacy of prevention and treatment programs.

The goal of this dissertation is to contribute empirical evidence regarding modifiable individual and social factors that a person can address to change their IPV victimization

trajectories and work towards intentional relational well-being. This research utilizes a syndemics framework (Singer, 2009; Singer, Bulled, Ostrach, & Mendenhall, 2017; Singer & Clair, 2003) to identify the trauma exposure, mental health, and sociodemographic contributions to IPV victimization risk across three independent studies in order to address the following three specific aims: 1) to identify the contributions of trauma exposure, mental health, and sociodemographic factors to total amount of IPV victimization in women with children over an eight-year period; 2) to prospectively examine the trauma exposure, mental health, and sociodemographic factors that contribute risk for experiencing IPV re-engagement in women with children across eight years; 3) to delineate the independent and cumulative roles of trauma exposure and mental health concerns on odds of experiencing IPV victimization in a community sample of emerging adults recruited from an urban emergency department. Taken together, these studies extend the current body of evidence on the confluence of risk factors for experiencing IPV victimization with the ultimate goal to inform novel intervention targets for the mitigation of IPV victimization.

The Dissertation Studies

Study 1

Although much remains unknown about what creates risk for women's IPV victimization across time, trauma exposure and mental health have been identified as likely contributors. Specifically, posttraumatic stress (PTS) has been robustly linked to IPV victimization, yet little is understood about the unique contributions of PTS symptom domains to IPV risk. Identification of PTS symptoms that confer risk for IPV has the potential to inform novel targets of intervention to improve the precision of current treatments that show limited effectiveness at reducing IPV victimization. This study follows women with children ($N = 118$) across eight

years to identify the trauma exposure, mental health, and sociodemographic factors that contribute to IPV victimization risk using a longitudinal OLS regression.

Outcomes of study one revealed that higher levels of PTS symptoms were associated with initially greater IPV victimization. However, across time women with higher PTS symptoms decreased more quickly in the amount of IPV victimization than those with lower PTS symptoms. Follow-up analyses revealed that higher levels of PTS arousal and reexperiencing were each associated with initially higher levels of IPV victimization. However, over time women with high levels of PTS arousal decreased more quickly in the amount of IPV victimization than those with low levels of PTS arousal. This was not the case for reexperiencing: higher levels of PTS reexperiencing started off and remained associated with higher levels of IPV victimization across time. Women's age was inversely related to IPV victimization over time only when accounting for the PTS symptom domains. Findings suggest that collapsing PTS symptoms into an overall construct may be too imprecise to identify key mechanisms of IPV victimization risk. Intervention efforts should prioritize addressing reexperiencing symptoms as a pathway to limiting future IPV victimization.

Study 2

Although 35 - 56% of women with IPV victimization report experiencing victimization with more than one violent partner, few studies have identified factors that increase risk for experiencing IPV across multiple partners (i.e., IPV re-engagement). This study (N=120) follows women with IPV victimization over eight years to examine the role of trauma exposure, mental health, and socio-demographic factors in creating risk for re-engagement. Participants were drawn from a randomized control trial evaluating an intervention for mothers who had

experienced IPV. Multilevel modeling was used to examine trauma exposure, mental health, and sociodemographic risk factors of women's re-engagement over eight years.

Results revealed that more psychological but less sexual IPV was associated with greater re-engagement. Greater number of posttraumatic reexperiencing symptoms were associated with less re-engagement. Depressive symptoms were also significantly associated with re-engagement such that lower levels of positive affect and higher somatic symptoms were associated with higher re-engagement. Higher income and lower housing instability were associated with more re-engagement ($\beta_{\text{range}} = -.125 - .160$). Finally, participation in the intervention program was significantly associated with lower levels of re-engagement at the eight-year follow-up compared to controls ($\beta = -0.747$, $p = .001$). Findings suggest that it is not what happened (i.e., experiences of abuse) that creates risk for re-engagement, but rather how a woman is doing following traumatic experiences (i.e., traumatic stress and depressive symptoms). Findings support the long-term effectiveness of a brief intervention in reducing re-engagement.

Study 3

Emerging adulthood (ages 18 to 25) is a unique developmental period (Arnett, 2000) when IPV involvement may peak or solidify (Rennison & Rand, 2003; White et al., 2009) as this is a time when intimate relationships become more serious (Arnett, 2000) and substance use also peaks (Jackson et al., 2008; White et al., 2009). IPV involvement during this period is associated with catastrophic social and personal costs that may extend throughout the lifetime (Liebschutz & Rothman, 2012). Yet, little is understood about what confers risk for experiencing IPV victimization in emerging adulthood, especially outside of samples of college students (Coker et al., 2016). The third study examines emerging adults ($N = 645$) who received services for any reason at a Level 1 urban emergency department to delineate the independent and cumulative

roles of trauma exposure (non-partner violence (NPV) victimization) and mental health concerns (anxiety, IPV aggression, alcohol use, and marijuana use) on the odds of experiencing IPV victimization while controlling for known sociodemographic contributors of risk.

Study three findings suggest that trauma exposure (NPV victimization) was not significantly associated with IPV victimization, nor were the mental health concerns of anxiety or marijuana use alone. However, IPV aggression, alcohol consumption, and identification as female compared to male were each associated with increased IPV victimization risk. Evidence was not found for a syndemic relationship between trauma exposure and mental health. However, findings suggested a synergistic relationship between IPV aggression and alcohol use and alcohol use and marijuana use on IPV victimization risk. At low levels of IPV aggression (1-3 acts in the prior six months), alcohol use was associated with increased IPV victimization risk, but this did not hold for four acts or more. Here, IPV victimization risk remained high across all levels of alcohol consumption. Results also showed that those who did not use marijuana had significantly higher IPV victimization risk as alcohol use increased. However, this was not true of those who did use marijuana as IPV victimization risk did not increase as alcohol use increased. Current findings suggest that alcohol use is an important but insufficient predictor of IPV victimization risk alone but needs to be considered in tandem with other important indicators of risk.

Conclusions

Taken together, these three studies add to the field through identifying factors that those who have experienced IPV victimization can influence to affect their future IPV victimization risk, namely through addressing current mental health concerns including PTS and depressive symptoms as well as substance use. Prior evidence suggests that IPV is a dyadic and relational

process, meaning that characteristics of both the person perpetrating and the person experiencing the IPV victimization may contribute risk to the occurrence of violence between intimate partners (Kuijpers, Van der Knaap, et al., 2012b; Moffitt et al., 2001). This dissertation research directly addresses the call in the extant literature to identify factors that those experiencing IPV victimization can influence to reduce future risk for victimization (Goodman et al., 2005; Kuijpers et al., 2011; Perez & Johnson, 2008).

Findings from this research highlight the limitations of using overall mental health diagnostic constructs when examining the mechanisms of risk for IPV victimization. Diagnostic categories such as PTS and depression are comprised of disparate symptoms (Foa et al., 1995; Schauer & Elbert, 2010) that may pose differential risk or protection for IPV victimization. Evidence from the current works thus reveals that it is critical to examine mental health concerns at least at the symptom domain level when delineating mechanisms of risk for IPV victimization. Larger diagnostic constructs such as PTS, depression, and anxiety may just be too imprecise to adequately capture contributors of risk.

Results from the three studies also suggest that it is not so much “what happened to you” (i.e., experiences of abuse) that create risk for IPV victimization rather it is “how you are doing” (i.e. your functioning - mental health concerns including substance use) that matters. Even though “what happened to you” and “how you are doing” are inextricably linked, this is an important finding as it suggests that there are modifiable factors that an individual can influence to affect their future IPV victimization risk including addressing current the mental health concerns of PTS arousal and reexperiencing and the depressive symptoms of positive affect and somatic concerns, IPV aggression, and alcohol use. Prior work highlighting the impact of Adverse Childhood Experiences (Whitfield et al., 2003) on IPV victimization risk may suggest

that prior experiences were the main contributor to risk without identifying a pathway for healing. The present work helps to illuminate that past experiences matter as they may be linked to our current functioning, but it is the latter that is directly linked to IPV victimization risk. This finding is hopeful as there are many effective treatment options to support improvement in functioning.

Finally, the three dissertation studies provide important evidence of the complex nature of the mechanism of risk for IPV victimization. Studies one and two not only revealed the importance of examining mental health concerns at the symptom-level rather than as overall diagnostic constructs, but study three also revealed the importance of alcohol use as a risk factor only when considered as an interaction with other potential contributors of risk (i.e. IPV aggression and marijuana use). These findings suggest that the study of mechanisms of IPV victimization risk must adequately consider the complexity of human experience and the cumulative impact of several factors in an individual's environment on overall risk.

Clinical Implications

Given the novel nature of this work and the below described limitations, it is too early to suggest robust clinical implications of the current findings. Even so, results have promising potential clinical value as specific PTS and depression symptom domains were identified as likely contributors to IPV victimization risk. Identification of these specific mental health concerns could serve to both narrow the scope and precisely focus treatment. Thus, continued research may have the capacity to vastly improve IPV victimization treatment outcomes through increasing the precision of the mechanisms targeted through intervention. Clinicians could screen for identified modifiable risk factors for victimization and tailor treatment to address the vulnerabilities present in each case.

Limitations

Studies one and two were influenced by issues of missing data due to participant attrition across the eight years of the study: of the 120 women interviewed at baseline, 71 were retained at time three (six to nine months later) and 68 at time four (eight years later). Although no significant differences were identified between the women retained and those lost to attrition on any study variables of interest, it is likely that the data was not completely missing at random. Specifically, in study two it is likely that the outcome of re-engagement at time 4 was not missing at random, particularly in the treatment group, as the mean for re-engagement for this group at time 4 is lower than time 3. Future studies would benefit from additional contact with participants between data collection waves to limit attrition. In addition, study measurement occasions were unequally distributed across time, which may have contributed to more unreliable retrospective reporting on some aspects of IPV victimization at some time points compared to others (e.g. on re-engagement at time four compared to re-engagement at time three). Although long-term longitudinal research is urgently needed to continue to prospectively delineate the mechanisms of risk for IPV victimization, future work should consider adding in additional assessment opportunities that are equally spaced throughout the duration of the study to provide more comprehensive longitudinal data and reduce statistical noise.

The three dissertation studies each examined risk factors for three different aspects of IPV victimization: study one examined the total amount of IPV victimization experienced, study two, IPV re-engagement (the number of violent partners), and study three, the odds of experiencing IPV victimization. Although this research provides novel insight into the mechanisms of IPV victimization risk, the assessment of IPV in these studies is not without limitations. Study two examined women's IPV re-engagement but relied on a single question to

assess the number of violent partners. Variation in responses on this item across waves contributed to increased statistical noise and greater difficulty determining the true nature of the observed relationships. Future work needs to focus on the development of a comprehensive assessment tool with greater reliability for assessing re-engagement. The three studies also did not include any empirical assessment of information about the partners or relationships including the length of the relationship, the amount and type of IPV during the relationship, any contextual factors, or any information on reasons for staying with or barriers to leaving a violent partner. Further research should utilize assessments to comprehensively capture the nature and contextual factors associated with IPV. In addition, findings from study three suggest that IPV perpetration is an important risk factor for of IPV victimization among emerging adults, independent of sex, which coincides with prior empirical evidence (Baker & Stith, 2008; Renner & Whitney, 2012). Studies one and two were limited, however, in that women were only assessed for unidirectional IPV (only IPV victimization was assessed) as the research was done in community settings that limited the types of assessments allowed. Given strong evidence that the majority of IPV is bidirectional (i.e. both IPV victimization and aggression; Cunradi et al., 2020; Renner & Whitney, 2012), future work would benefit from including assessments of both IPV victimization and aggression when possible to continue to comprehensively delineate the mechanisms of risk for IPV victimization.

Study three was a secondary analysis of cross-sectional data that was collected as part of a National Institute of Health K01 grant to develop an online mindfulness-based cognitive behavioral and motivational interviewing intervention to reduce IPV perpetration and co-occurring alcohol use. Although findings from this third study provide important insight into the contributing mechanisms for odds of IPV victimization in emerging adults, the use of secondary

data was a limitation to the work. First, available data was cross-sectional, which precludes the drawing of causal inferences. Future work should focus on prospective longitudinal designs that would allow for the identification of potential causal mechanisms for IPV victimization risk in this population. Second, given that recruitment for the current study was done in the ED, the length of the survey administered to participants had to be limited to accommodate the demands of the hospital. Future research would benefit from longer interviews to facilitate the administration of more complete standardized measures, specifically for mental health concerns. This is especially important as the findings from studies one and two of this dissertation revealed that mental health concerns conceptualized as disorders or total constructs (e.g. depression) rather than symptom domains (e.g. somatic symptoms) were insufficient to fully delineate IPV victimization risk. Continued work is needed comprehensively assesses mental health concerns at the symptom level. Third, study three did not include a measure of posttraumatic stress, which would be vital to include in future research given its established associations with IPV involvement both in this dissertation and elsewhere in the literature (Dutton et al., 2006; Ehrensaft, Moffitt, & Caspi, 2006; Golding, 1999).

This dissertation utilized a syndemics framework (Singer, 2009; Singer, Bulled, Ostrach, & Mendenhall, 2017; Singer & Clair, 2003) to examine how trauma exposure and mental health concerns may overlap and mutually contribute to one another leading to increased IPV victimization. Central to the theory of syndemics is the idea that the social contexts in which these interactions occur fundamentally contribute and exacerbate how the epidemics interact, thus sociodemographic were further considered with trauma exposure and mental health in their contributions to IPV victimization. Although this is a comprehensive and novel theoretical framework for examining the mechanisms of IPV victimization risk, the current studies were

limited in the methodological application of the framework due to issues of statistical power and the operationalization of trauma exposure and mental health constructs. Namely, though study three examined the interaction between trauma exposure and each of the included mental health concerns, studies one and two were unable to examine such direct interactions due to issues of power resulting from a limited sample size. Recent review articles have cited this lack of examination of the interaction of disease clusters as a widespread issue with methodological applications of the framework in extant research (Merrill Singer et al., 2020; Tsai et al., 2017). Findings from these reviews suggests that the majority of using a syndemics framework addresses only issues of mutual causality/co-occurrence of diseases rather than the interaction of disease clusters due to a lack of examination of interactions in analyses (Tsai et al., 2017). Thus, although the current dissertation studies provide novel evidence on the potentially mutually causal roles of trauma exposure and mental health on IPV victimization, future research needs to utilize large enough samples to be able to fully explore the potential interaction between said disease clusters on IPV victimization risk. In addition, the current work may be limited by an overly broad conceptualization of the disease clusters of trauma exposure and mental health. Findings from studies one and two revealed that very specific aspects of mental health (e.g. PTS reexperiencing) and trauma exposure (e.g. psychological IPV victimization) were each associated with IPV victimization risk. Thus, trauma exposure and mental health concerns may be too imprecise as overall constructs with too much individual variability to be useful in this framework. Future research should more precisely consider the interaction between the symptom domains of PTS and past psychological IPV victimization as having a potential syndemic relationship with IPV victimization given current findings.

Future directions

The present work represents a strong first step in identifying modifiable factors that an individual can address to affect their IPV victimization risk and thus increase their potential for relational well-being with intimate partners. Additionally, these findings reveal several new lines of inquiry for future direction of study for mechanisms of IPV victimization risk including larger questions around the relevance of developmental timing of trauma exposure, the measurement and operationalization of PTS, the examination of relational fear and attachment that may be associated with increased risk of relational turmoil and IPV victimization, and the study of the efficacy of additional relationally-focused modalities of intervention that target symptoms of Complex Posttraumatic Stress Disorder such as Internal Family Systems Therapy (Schwartz, 2013), Sensorimotor Psychotherapy (Ogden & Fisher, 2015), and Trauma-Informed Stabilization (Fisher, 2017b) .

Developmental timing of trauma exposure

The current studies examined the contribution of trauma exposures that occurred over the lifespan to IPV victimization risk, though largely without accounting for the timing of the exposures due to data availability. Yet, a large body of work has clearly delineated the importance of the timing of traumatic exposure on negative outcomes throughout the lifetime (Bosch et al., 2012). Particularly, evidence has demonstrated that traumatic exposures early in development have the most harmful impacts throughout the lifespan (Felitti et al., 1998), though this is not well understood for IPV victimization risk. Future research would benefit from more comprehensive assessment of traumatic exposures throughout the lifespan that includes a clear delineation of the developmental timing of the exposures. Although promising new work is examining the timing of maternal trauma exposure on child outcomes (Prenatal Stress Study; Levendosky, Bogat, Lonstein, & Muzik, 2016; 2020; R01HD100469, R01HD085990), to date

limited research has examined the role of the timing of past trauma exposures on IPV victimization risk. Elucidating the developmental periods at which traumatic injuries occurred that are associated with future IPV victimization risk could inform novel intervention targets by providing information regarding the likely missing emotional and relational experiences associated with the traumatic injury and the developmental needs that may be essential to address to support real healing from these traumatic experiences even in adulthood.

Measurement and operationalization of PTS

Studies one and two revealed the importance of PTS on IPV victimization risk, specifically delineating the role of reexperiencing symptoms for risk. Given this evidence, future work would benefit from using more comprehensive clinical interview assessments of PTS than were available for the current research. Although standardized measures are widely used in research in psychology such as the Posttraumatic Diagnostic Scale (Foa et al., 1997) used in studies one and two, clinical interviews conducted by trained clinicians are thought to be the gold standard for capturing symptoms. This may be particularly important in the case of PTS as a well-trained, trauma-informed clinician may be able to identify PTS reexperiencing symptoms more readily, for example, in a trauma narrative and ask the necessary follow-up questions for confirmation than a participant themselves without any framework or psychoeducation to contextualize their lived experience following traumatic experiences. Given the likely role of PTS in IPV victimization risk, the comprehensive and accurate characterization of PTS symptoms is a crucial future direction to explicate the true mechanisms of risk for IPV victimization.

Future research may also benefit from expanding the construct of PTS beyond the DSM diagnostic category of Posttraumatic Stress Disorder (PTSD) to consider conceptualizations of

complex PTSD (C-PTSD; Herman, 1992) that recognize the cumulative toll of adversity and trauma on symptom complexity (M. Cloitre et al., 2009). Given study one of the current research revealed the limitations of using PTS as an overall construct, consideration of C-PTSD may be an important avenue of study as it considers both the extent and complexity of symptom presentations (Herman, 1992). Furthermore, the inclusion of C-PTSD would allow for more nuanced consideration of the relational impact and the effects on identify of early adversity, which is a potentially important and understudied area of inquiry for delineating IPV victimization risk.

Likewise, another valuable avenue for future study would be to comprehensively consider the role of dissociation in IPV victimization risk. Given that dissociation is a feature of PTS reexperiencing (American Psychiatric Association, 2013) - identified in this dissertation research as a key mechanisms for IPV victimization risk - greater attention is needed to understand the role of altered states of consciousness for risk of this violence. Specifically, it may be that individuals that have more dissociative internal fragmentation resulting from early traumatic experiences (Fisher, 2017) may be a greater risk for experiencing relational violence than those without such fragmentation. Identification of the relevance of this dissociation for IPV victimization could be invaluable as there are several promising treatments to support healing from this type of early splitting resulting from traumatic exposures (Fisher, 2017b, 2017a; Schwartz, 2013).

Relational fear and attachment

Given the potential role of the developmental timing of traumatic exposures and hypothesized importance of C-PTSD in IPV victimization risk, future studies may also benefit from assessing participants for relational fear and attachment styles. Prior work has identified

that early childhood experiences of unsolvable fear are linked to diminished capacity for managing threat in adulthood, though the causal mechanisms of this association have yet to be identified (Cassidy & Mohr, 2001). Targeted work assessing key cognitions and feeling states that may contribute risk for IPV victimization could illuminate invaluable new targets of intervention. Thus, the inclusion of additional measures in future research such as the Experiences in Close Relationship Scale (Brennan et al., 1998), for example, could provide previously unexamined information on the potential role of relational fear in IPV victimization risk.

Testing additional relationally focused modalities of intervention

Study two revealed the efficacy of the Moms Empowerment Program (MEP; Graham-Bermann, 2011) at reducing IPV re-engagement over eight years, which joins a wealth of additional evidence for the effectiveness of the treatment for improving parenting (Grogan-Kaylor et al., 2016; Howell et al., 2015), depression (Sara F. Stein et al., 2018), PTSD (Galano et al., 2016; S. A. Graham-Bermann & Miller, 2013), physical health (Hannah M. Clark et al., 2018), and short term IPV victimization (over 6-9 months; Miller et al., 2014) in IPV-exposed women. However, evidence for the effectiveness of the intervention at reducing total amount of IPV victimization across eight years was not found in study one suggesting that continued adaptations and alterations to treatment are needed to address IPV victimization across longer spans of time. Even though the long-term goal of this research is to identify the mechanisms of risk for IPV victimization to inform novel targets of intervention, it may be that some extant treatments already target the identified and hypothesized mechanisms of IPV victimization risk given their specific focus on healing C-PTSD. Thus, future work should also consider testing the efficacy of additional relationally-focused modalities of interventions that target addressing C-

PTSD including Internal Family Systems Therapy (Schwartz, 2013), Sensorimotor Psychotherapy (Ogden & Fisher, 2015), and Trauma-Informed Stabilization (Fisher, 2017b, 2017a). Although these interventions show promise for sustainable healing from C-PTSD, which may be an important mechanism of IPV victimization risk, no rigorous randomized control trials have specifically examined the efficacy of these modalities at reducing IPV victimization.

Summary

The aim of this dissertation research was to add to the limited body of knowledge of risk factors for IPV victimization to inform the development and refinement of intervention programs indicated for IPV reduction and mitigation. This is a particularly needed area of focus as current treatments for those who have experienced this violence have found limited support for their effectiveness in reducing future IPV victimization (Eckhardt et al., 2013), raising questions about the appropriateness of the chosen mechanisms targeted through these intervention programs. This dissertation has made significant contributions to field through identifying factors that those who have experienced IPV victimization can influence to affect their future IPV victimization risk. Evidence from the current works thus reveals that it is critical to examine mental health concerns at least at the symptom domain level when delineating mechanisms of risk for IPV victimization. Larger diagnostic constructs may just be too imprecise to adequately capture contributors of risk. Results also suggest that it is not so much “what happened to you” (i.e., experiences of abuse) that create risk for IPV victimization rather it is “how you are doing” (i.e. your functioning - mental health concerns including substance use) that matters. Finally, findings indicate that the study of mechanisms of IPV victimization risk must adequately consider the complexity of human experience and the cumulative impact of several factors in an individual’s environment on overall risk. Continued research is needed to comprehensively delineate precise

and actionable mechanisms of risk for IPV victimization to inform novel intervention targets and improve the efficacy of prevention and treatment programs for IPV victimization mitigation and to support individuals towards living in intentional relational well-being.

References

- Afifi, T. O., Henriksen, C. A., Asmundson, G. J. G., & Sareen, J. (2012). Victimization and perpetration of intimate partner violence and substance use disorders in a nationally representative sample. *Journal of Nervous and Mental Disease*, 200, 684–691.
DOI:10.1097/NMD.0b013e3182613f64
- Ahmadabadi, Z., Najman, J. M., Williams, G., & Clavarino, A. M. (2017). Income, gender, and forms of intimate partner violence. *Journal of Interpersonal Violence*, 1–26.
DOI:10.1177/0886260517719541
- Alexander, P. C. (2009). Childhood trauma, attachment, and abuse by multiple partners. *Psychological Trauma: Theory, Research, Practice, and Policy*, 1, 78–88.
DOI:10.1037/a0015254
- American Psychiatric Association. (2000). Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR). In *American Psychiatric Publication: Vol. Text Revis* (Fourth Edi). DOI:10.1001/jama.1994.03520100096046
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*. American Psychiatric Pub.
- American Psychiatric Association, & APA, A. P. A. (2013). Diagnostic and Statistical Manual of Mental Disorders: DSM-5. In *American Psychiatric Publication: Vol. Text Revis* (Fourth Edi). American Psychiatric Association. DOI:10.1001/jama.1994.03520100096046
- Anderson, K. L. (2002). Perpetrator or victim? Relationships between intimate partner violence and well-being. *Journal of Marriage and Family*, 64, 851–863. DOI:10.1111/j.1741-

3737.2002.00851.x

- Arnett, J. J. (2000). Emerging adulthood: A theory of development from the late teens through the twenties. *American Psychologist*, 55, 469–480. DOI:10.1037//0003-066X.55.5.469
- Azziz-Baumgartner, E., McKeown, L., Melvin, P., Dang, Q., & Reed, J. (2011). Rates of femicide in women of different races, ethnicities, and places of birth: Massachusetts, 1993–2007. *Journal of Interpersonal Violence*, 26, 1077–1090.
<http://jiv.sagepub.com/cgi/doi/10.1177/0886260510365856>
- Babcock, J. C., Green, C. E., & Robie, C. (2004). Does batterers' treatment work? A meta-analytic review of domestic violence treatment. *Clinical Psychology Review*, 23, 1023–1053. DOI:10.1016/j.cpr.2002.07.001
- Baker, C. R., & Stith, S. M. (2008). Factors Predicting Dating Violence Perpetration Among Male and Female College Students. *Journal of Aggression, Maltreatment & Trauma*, 17, 227–244. DOI:10.1080/10926770802344836
- Bandura, A. (1978). The self system in reciprocal determinism. *American Psychologist*, 33, 344–358. DOI:10.1037/0003-066X.33.4.344
- Barrios, Y. V., Gelaye, B., Zhong, Q., Nicolaidis, C., Rondon, M. B., Garcia, P. J., Sanchez, P. A. M., Sanchez, S. E., & Williams, M. A. (2015). Association of Childhood Physical and Sexual Abuse with Intimate Partner Violence, Poor General Health and Depressive Symptoms among Pregnant Women. *Plos One*, 10. /10.1371/journal.pone.0116609
- Becker, K. D., Stuewig, J., & McCloskey, L. A. (2010). Traumatic Stress Symptoms of Women Exposed to Different Forms of Childhood Victimization and Intimate Partner Violence. *Journal of Interpersonal Violence*, 25, 1699–1715. DOI:10.1177/0886260509354578
- Bell, K. M., & Naugle, A. E. (2014). Intimate partner violence theoretical considerations:

- Moving towards a contextual framework. *Clinical Psychology Review*, 28, 1096–1107.
DOI:10.1016/j.cpr.2008.03.003
- Bell, M. E., Cattaneo, L. B., Goodman, L. A., & Dutton, M. A. (2008). Assessing the risk of future psychological abuse: Predicting the accuracy of battered women's predictions. *Journal of Family Violence*, 23, 69–80. DOI:10.1007/s10896-007-9128-5
- Berzenski, S. R., & Yates, T. M. (2010). A Developmental Process Analysis of the Contribution of Childhood Emotional Abuse to Relationship Violence. *Journal of Aggression, Maltreatment & Trauma*, 19, 180–203. DOI:10.1080/10926770903539474
- Beydoun, H. A., Beydoun, M. A., Kaufman, J. S., Lo, B., & Zonderman, A. B. (2012). Intimate partner violence against adult women and its association with major depressive disorder, depressive symptoms and postpartum depression: systematic review and meta-analysis. *Social Science & Medicine*, 75, 959–975. DOI:10.1177/0022146513520430
- Black, M. C., Basile, K. C., Breiding, M. J., Smith, S. G., Walters, M. L., Merrick, M. T., Chen, J., & Steven, M. R. (2011). *National Intimate Partner and Sexual Violence Survey (NISVS): 2010 summary report*.
- Blasco-Ros, C., Sánchez-Lorente, S., & Martinez, M. (2010). Recovery from depressive symptoms, state anxiety and post-traumatic stress disorder in women exposed to physical and psychological, but not to. *BMC Psychiatry*. DOI:10.1186/1471-244X-10-98
- Bliton, C. F., Wolford-Clevenger, C., Zapor, H., Elmquist, J. A., Brem, M. J., Shorey, R. C., & Stuart, G. L. (2016). Emotion Dysregulation, Gender, and Intimate Partner Violence Perpetration: An Exploratory Study in College Students. *Journal of Family Violence*, 31, 371–377. DOI:10.1007/s10896-015-9772-0
- Bogat, G. A., Levendosky, A. A., Theran, S., Von Eye, A., & Davidson, W. S. (2003). Predicting

- the Psychosocial Effects of Interpersonal Partner Violence (IPV): How much does a Woman's History of IPV Matter? *Journal of Interpersonal Violence*, 18, 1271–1291.
DOI:10.1177/0886260503256657
- Bonomi, A.E., Thompson, R. S., Anderson, M., Reid, R. J., Carrell, D., Dimer, J. A., & Rivara, F. P. (2006). Intimate partner violence and women's physical, mental, and social functioning. *American Journal of Preventive Medicine*, 30, 458–466.
- Bonomi, Amy E., Anderson, M. L., Cannon, E. A., Slesnick, N., & Rodriguez, M. A. (2009). Intimate partner violence in Latina and non-Latina Women. *American Journal of Preventive Medicine*, 36, 43–48. DOI:10.1016/j.amepre.2008.09.027
- Bosch, N. M., Riese, H., Reijneveld, S. A., Bakker, M. P., Verhulst, F. C., Ormel, J., & Oldehinkel, A. J. (2012). Timing matters: Long term effects of adversities from prenatal period up to adolescence on adolescents' cortisol stress response. The TRAILS study. *Psychoneuroendocrinology*, 37, 1439–1447. DOI:10.1016/j.psyneuen.2012.01.013
- Bowling, J. M. (2007). Correlates of Intimate Partner Violence Among Female. *North Carolina Medical Journal*, 68, 89–94.
<http://jiv.sagepub.com/cgi/content/abstract/19/12/1494%5Cnhttp://jiv.sagepub.com/content/19/12/1494.full.pdf>
- Bradley, K. A., Debenedetti, A. F., Volk, R. J., Williams, E. C., Frank, D., & Kivlahan, D. R. (2007). AUDIT-C as a brief screen for alcohol misuse in primary care. *Alcoholism: Clinical and Experimental Research*, 31, 1208–1217. DOI:10.1111/j.1530-0277.2007.00403.x
- Breiding, M., Chen, J., & Black, M. (2014). *Intimate Partner Violence in the United States — 2010*.
- Breiding, M. J., Black, M. C., & Ryan, G. W. (2008). Prevalence and Risk Factors of Intimate

- Partner Violence in Eighteen U.S. States/Territories, 2005. *American Journal of Preventive Medicine*, 34, 112–118. DOI:10.1016/j.amepre.2007.10.001
- Breiding, M. J., Smith, S. G., Basile, K. C., Walters, M. L., Chen, J., & Merrick, M. T. (2014). Prevalence and characteristics of sexual violence, stalking, and intimate partner violence victimization - National Intimate Partner and Sexual Violence Survey, United States, 2011. *Morbidity and Mortality Weekly Report. Surveillance Summaries*, 63, 1–18.
<http://vb3lk7eb4t.search.serialssolutions.com.libproxy.lib.unc.edu/?sid=Entrez:PubMed&id=pmid:25188037>
- Brennan, K. A., Clark, C. L., & Shaver, P. R. (1998). Self-report measurement of adult attachment: An integrative overview. In J. A. Simpson & W. S. Rholes (Eds.), *Attachment theory and close relationships* (pp. 44–76). Guilford Press.
- Brenner, I., & Ben-Amitay, G. (2015). Sexual revictimization: The impact of attachment anxiety, accumulated trauma, and response to childhood sexual abuse disclosure. *Violence and Victims*, 30, 49–65. DOI:10.1891/0886-6708.VV-D-13-00098
- Brezing, C., Ferrara, M., & Freudenreich, O. (2015). The syndemic illness of HIV and trauma: Implications for a trauma-informed model of care. *Psychosomatics*, 56, 107–118.
DOI:10.1016/j.psych.2014.10.006
- Briere, J., & Runtz, M. (1987). Post sexual abuse trauma: Data and implications for clinical practice. *Journal of Interpersonal Violence*, 2, 367–379.
DOI:10.1177/088626058700200403
- Bush, K., Kivlahan, D. R., McDonnell, M. B., Fihn, S. D., & Bradley, K. A. (1998). The AUDIT Alcohol Consumption Questions (AUDIT-C): An effective brief screening test for problem drinking. *Archives of Internal Medicine*, 158, 1789–1795.

- Bushman, B. J., & Cooper, H. M. (1990). Effects of alcohol on human aggression: an integrative research review. *Psychological Bulletin*, 107, 341–354. DOI:10.1037/0033-2909.107.3.341
- Caetano, R., Field, C. a, Ramisetty-Mikler, S., & McGrath, C. (2005). The 5-year course of intimate partner violence among White, Black, and Hispanic couples in the United States. *Journal of Interpersonal Violence*, 20, 1039–1057. DOI:10.1177/0886260505277783
- Caetano, R., McGrath, C., Ramisetty-Mikler, S., & Field, C. A. (2005). Drinking, Alcohol Problems and the Five-Year Recurrence and Incidence of Male to Female and Female to Male Partner Violence. *Alcoholism: Clinical and Experimental Research*, 29, 98–106. DOI:10.1097/01.ALC.0000150015.84381.63
- Caetano, R., Schafer, J., & Cunradi, C. B. (2001). Alcohol-related intimate partner violence among White, Black, and Hispanic couples in the United States. *Alcohol Res Health*, 25, 58–65.
- Caetano, R., Vaeth, P. A. C., & Ramisetty-Mikler, S. (2008). Intimate partner violence victim and perpetrator characteristics among couples in the United States. *Journal of Family Violence*, 23, 507–518. DOI:10.1007/s10896-008-9178-3
- Caldwell, J. E., Swan, S. C., & Woodbrown, V. D. (2012). Gender differences in intimate partner violence outcomes. *Psychology of Violence*, 2, 42–57. DOI:10.1037/a0026296
- Cano, A., & Vivian, D. (2003). Are Life Stressors Associated with Marital Violence? *Journal of Family Psychology*, 17, 302–314. DOI:10.1037/0893-3200.17.3.302
- Capaldi, D. M., & Kim, H. K. (2007). Typological approaches to violence in couples: A critique and alternative conceptual approach. *Clinical Psychology Review*, 27, 253–265. DOI:10.1016/j.cpr.2006.09.001
- Capaldi, D. M., Knoble, N. B., Shortt, J. W., & Kim, H. K. (2012). A Systematic Review of Risk

Factors for Intimate Partner Violence. *Partner Abuse*, 3, 231–280. DOI:10.1891/1946-6560.3.2.231.A

Capaldi, D. M., Shortt, J. W., & Kim, H. K. (2005). A life span developmental systems perspective on aggression toward a partner. In W. M. Pinsof & J. L. Lebow (Eds.), *Oxford series in clinical psychology. Family psychology: The art of the science* (pp. 141–167). Oxford University Press.

Cassidy, J., & Mohr, J. J. (2001). Unsolvable Fear, Trauma, and Psychopathology: Theory, Research, and Clinical Considerations Related to Disorganized Attachment Across the Life Span. *Clinical Psychology: Science and Practice*, 8, 275–298. DOI:10.1093/clipsy.8.3.275

Cattaneo, L. B., & Goodman, L. A. (2005). Risk Factors for Reabuse in Intimate Partner Violence: A Cross-Disciplinary Critical Review. *Trauma, Violence, & Abuse*, 6, 141–175. DOI:10.1177/1524838005275088

Champely. (2018). *Pwr: Basic functions for power analysis*.

Chandan, J. S., Thomas, T., Bradbury-Jones, C., Russell, R., Bandyopadhyay, S., Nirantharakumar, K., & Taylor, J. (2019). Female survivors of intimate partner violence and risk of depression, anxiety and serious mental illness. *The British Journal of Psychiatry*, 1–6. DOI:10.1192/bjp.2019.124

Chapman, H., & Gillespie, S. M. (2019). The Revised Conflict Tactics Scales (CTS2): A review of the properties, reliability, and validity of the CTS2 as a measure of partner abuse in community and clinical samples. *Aggression and Violent Behavior*, 44, 27–35. DOI:10.1016/j.avb.2018.10.006

Chermack, S. T., Fuller, B. E., & Blow, F. C. (2000). Predictors of expressed partner and non-partner violence among patients in substance abuse treatment. *Drug and Alcohol*

- Dependence*, 58, 43–54. DOI:10.1016/S0376-8716(99)00067-8
- Chermack, S. T., & Giancola, P. R. (1997). The relation between alcohol and aggression: An integrated biopsychosocial conceptualization. *Clinical Psychology Review*, 17, 621–649. DOI:10.1016/S0272-7358(97)00038-X
- Cho, H. (2012). Racial differences in the prevalence of intimate partner violence against women and associated factors. *Journal of Interpersonal Violence*, 27, 344–363. DOI:10.1177/0886260511416469
- Clark, H. M., Galano, M. M., Grogan-Kaylor, A. C., Montalvo-Liendo, N., & Graham-Bermann, S. A. (2016). Ethnoracial variation in women’s exposure to intimate partner violence. *Journal of Interpersonal Violence*, 31, 531–552. DOI:10.1177/0886260514555871
- Clark, Hannah M., Grogan-Kaylor, A. C., Galano, M. M., Stein, S. F., & Graham-Bermann, S. A. (2018). Moms’ Empowerment Program participation associated with improved physical health among Latinas experiencing intimate partner violence. *Revista Panamericana de Salud Publica/Pan American Journal of Public Health*, 42, 1–7. DOI:10.26633/rpsp.2018.39
- Cloitre, M., Stolbach, B. C., Herman, J. L., Kolk, B. V. D., Pynoos, R., Wang, J., & Petkova, E. (2009). A developmental approach to complex PTSD: Childhood and adult cumulative trauma as predictors of symptom complexity. *Journal of Traumatic Stress*, 22, 399–408. DOI:10.1002/jts
- Cloitre, Marylene, Tardiff, K., Marzuk, P. M., Leon, A. C., & Portera, L. (1996). Childhood abuse and subsequent sexual assault among female inpatients. *Journal of Traumatic Stress*, 9, 473–482. DOI:10.1007/BF02103659
- Cohen, J. (1988). *Statistical Power Analysis for the Behavioural Science* (2nd ed.).

- Coid, J., Petruckevitch, A., Feder, G., Chung, W., Richardson, J., & Moorey, S. (2001). Relation between childhood sexual and physical abuse and risk of revictimisation in women: a cross-sectional survey. *Lancet*, 358, 450–454. DOI:S0140673601056227 [pii]
- Coker, A. L., Follingstad, D. R., Bush, H. M., & Fisher, B. S. (2016). Are Interpersonal Violence Rates Higher Among Young Women in College Compared With Those Never Attending College? *Journal of Interpersonal Violence*, 31, 1413–1429.
DOI:10.1177/0886260514567958
- Cole, J., Logan, T., & Shannon, L. (2008). Women's Risk for Revictimization by a New Abusive Partner: For What Should We Be Looking? *Violence and Victims*, 23, 315–330.
DOI:10.1891/0886-6708.23.3.315
- Coolidge, F. L., & Anderson, L. W. (2002). Personality profiles of women in multiple abusive relationships. *Journal of Family Violence*, 17, 117–131. DOI:10.1023/A:1015005400141
- Cogle, J. R., Resnick, H., & Kilpatrick, D. G. (2009). A prospective examination of PTSD symptoms as risk factors for subsequent exposure to potentially traumatic events among women. *Journal of Abnormal Psychology*, 118, 405–411. DOI:10.1037/a0015370.A
- Crawford, E., & Wright, M. O. (2007). The Impact of Childhood Psychological Maltreatment on Interpersonal Schemas and Subsequent Experiences of Relationship Aggression. *Journal of Emotional Abuse*, 7, 93–116. DOI:10.1300/J135v07n02
- Cummings, A. M., Gonzalez-Guarda, R. M., & Sandoval, M. F. (2013). Intimate Partner Violence Among Hispanics: A Review of the Literature. *Journal of Family Violence*, 28, 153–171. DOI:10.1007/s10896-012-9478-5
- Cunradi, C. B., Caetano, R., & Schafer, J. (2002). Socioeconomic predictors of intimate partner violence among White, Black, and Hispanic couples in the United States. *Journal of Family*

- Violence*, 17, 601. DOI:0885-7482/02/1200-0377/0
- Cunradi, C. B., Dellor, E., Alter, H. J., Caetano, R., & Mair, C. (2020). Problem Drinking and Marijuana Use as Risks for Unidirectional and Bidirectional Partner Violence. *Partner Abuse*, 11, 57–75. DOI:10.1891/1946-6560.11.1.57
- Daigneault, I., Hébert, M., & McDuff, P. (2009). Men's and women's childhood sexual abuse and victimization in adult partner relationships: A study of risk factors. *Child Abuse and Neglect*, 33, 638–647. DOI:10.1016/j.chiabu.2009.04.003
- Dardis, C. M., Ullman, S. E., Rodriguez, L. M., Waterman, E. A., Dworkin, E. R., & Edwards, K. M. (2021). Bidirectional associations between alcohol use and intimate partner violence and sexual assault victimization among college women. *Addictive Behaviors*, 116, 106833. DOI:10.1016/j.addbeh.2021.106833
- Davies, L., Ford-Gilboe, M., Willson, A., Varcoe, C., Wuest, J., Campbell, J., & Scott-Storey, K. (2015). Patterns of cumulative abuse among female survivors of intimate partner violence: links to women's health and socioeconomic status. *Violence Against Women*, 21, 30–48. DOI:10.1007/s10896-012-9478-5
- Davies, Lorraine, Ford-gilboe, M., Willson, A., Varcoe, C., Wuest, J., Campbell, J., & Scott-Storey, K. (2015). Patterns of cumulative abuse among female survivors of intimate partner violence: Links to women's health and socioeconomic status. *Violence Against Women*, 21, 30–48. DOI:10.1177/1077801214564076
- Decker, M., & Littleton, H. L. (2018). Sexual Revictimization Among College Women: A Review Through an Ecological Lens. *Victims and Offenders*, 13, 558–588. DOI:10.1080/15564886.2017.1390514
- Derogatis, L. R. (2001). *Brief symptom inventory 18*. John Hopkins University.

- Derogatis, L. R., & Melisaratos, N. (1983). The Brief Symptom Inventory: an introductory report. *Psychological Medicine*, *13*, 595–605. DOI:10.1017/S0033291700048017
- Devries, K. M., Child, J. C., Bacchus, L. J., Mak, J., Falder, G., Graham, K., Watts, C., & Heise, L. (2014). Intimate partner violence victimization and alcohol consumption in women: A systematic review and meta-analysis. *Addiction*, *109*, 379–391. DOI:10.1111/add.12393
- Dobash, R. E., & Dobash, R. (1979). *Violence against wives: A case against the patriarchy*. The Free Press.
- Dutton, M. A. (2009). Pathways Linking Intimate Partner Violence and Posttraumatic Disorder. *Trauma, Violence, & Abuse*, *10*, 211–224. DOI:10.1177/1524838009334451
- Dutton, M. A., Green, B. L., Kaltman, S. I., Roesch, D. M., Zeffiro, T. A., & Krause, E. D. (2006). Intimate Partner Violence, PTSD, and Adverse Health Outcomes. *Journal of Interpersonal Violence*, *21*, 955–968. DOI:10.1177/0886260506289178
- Eckhardt, C. I., Murphy, C. M., Whitaker, D. J., Sprunger, J., Lafayette, W., & Woodard, K. (2013). The effectiveness of intervention programs for perpetrators and victims of intimate partner violence. *Partner Abuse*, *4*, 196–231. DOI:10.1891/1946-6560.4.2.196
- Ehrensaft, M. K., Moffitt, T. E., & Caspi, A. (2006). Is domestic violence followed by an increased risk of psychiatric disorders among women but not among men? A longitudinal cohort study. *American Journal of Psychiatry*, *163*, 885–892.
- Endler, N. S. (1997). Stress, anxiety and coping: The multidimensional interaction model. *Canadian Psychology*, *38*, 136–153. DOI:10.1037/0708-5591.38.3.136
- Epstein-Ngo, Q. M., Cunningham, R. M., Whiteside, L. K., Chermack, S. T., Booth, B. M., Zimmerman, M. A., & Walton, M. A. (2013). A daily calendar analysis of substance use and dating violence among high risk urban youth. *Drug and Alcohol Dependence*, *130*,

194–200. DOI:10.1016/j.drugalcdep.2012.11.006

- Epstein-Ngo, Q. M., Walton, M. A., Chermack, S. T., Blow, F. C., Zimmerman, M. A., & Cunningham, R. M. (2014). Event-level analysis of antecedents for youth violence: Comparison of dating violence with non-dating violence. *Addictive Behaviors*, 39, 350–353. DOI:10.1016/j.addbeh.2013.10.015
- Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., Koss, M. P., & Marks, J. S. (1998). Relationship of Childhood Abuse and Household Dysfunction to Many of the Leading Causes of Death in Adults: The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine*, 14, 245–258. DOI:10.1016/j.amepre.2019.04.001
- Field, C. A., & Caetano, R. (2003). Longitudinal model predicting partner violence among White, Black, and Hispanic couples in the United States. *Alcoholism: Clinical & Experimental Research*, 27, 1451–1458. <http://doi.wiley.com/10.1097/01.ALC.0000086066.70540.8C>
- Field, C. A., & Caetano, R. (2004). Ethnic differences in intimate partner violence in the U.S. general population: The role of alcohol use and socioeconomic status. *Trauma, Violence, & Abuse*, 5, 303–317. DOI:10.1177/1524838004269488
- Fisher, J. (2017a). *Healing the fragmented selves of trauma survivors: Overcoming internal self-alienation*. Taylor & Francis.
- Fisher, J. (2017b). Trauma-Informed Stabilisation Treatment: A New Approach to Treating Unsafe Behaviour. *Australian Clinical Psychologist*, 3, 55–62.
- Foa, E. B., Riggs, D. S., & Gershuny, B. S. (1995). Arousal, numbing, and intrusion: Symptom structure of PTSD following assault. *The American Journal of Psychiatry*, 152, 116–120.

DOI:<https://doi-org.proxy.lib.umich.edu/10.1176/ajp.152.1.116>

- Foa, Edna B., Cashman, L., Jaycox, L., & Perry, K. (1997). The validation of a self-report measure of posttraumatic stress disorder: The Posttraumatic Diagnostic Scale. *Psychological Assessment*, 9, 445–451. DOI:1040-3590/97/S3.00
- Foran, H. M., & Leary, K. D. O. (2008). Alcohol and intimate partner violence: A meta-analytic review. *Clinical Psychology Review*, 28, 1222–1234. DOI:10.1016/j.cpr.2008.05.001
- Fowler, D. (2007). The Extent of Substance Use Problems Among Women Partner Abuse Survivors Residing in a Domestic Violence Shelter. *Family & Community Health*, 30, S106–S108.
- Fritz, P. A. T., Slep, A. M. S., & O’Leary, K. D. (2012). Couple-level analysis of the relation between family-of-origin aggression and intimate partner violence. *Psychology of Violence*, 2, 139–153. DOI:10.1037/a0027370
- Galano, M. M., Grogan-Kaylor, A. C., Stein, S. F., Clark, H. M., & Graham-Bermann, S. A. (2016). Posttraumatic Stress Disorder in Latina Women: Examining the Efficacy of the Moms ’ Empowerment Program. *Psychological Trauma: Theory, Research, Practice, and Policy*. DOI:<http://dx.doi.org/10.1037/tra0000218>
- Gelles, R. J., & Straus, M. A. (1979). Determinants of violence in the family: Toward a theoretical integration. In W. et al Burr (Ed.), *Contemporary theories about the family*. Free Press.
- Gilbert, L., El-Bassel, N., Chang, M., Wu, E., & Roy, L. (2012). Substance use and partner violence among urban women seeking emergency care. *Psychology of Addictive Behaviors*, 26, 226–235. DOI:10.1037/a0025869
- Giles-Sims, J. (1983). *Wife battering: A systems theory approach*. Guilford Press.

- Golden, S. D., Perreira, K. M., & Durrance, C. P. (2013). Troubled times, troubled relationships: How economic resources, gender beliefs, and neighborhood disadvantage influence intimate partner violence. *Journal of Interpersonal Violence*, 28, 2134–2155.
<http://jiv.sagepub.com/cgi/doi/10.1177/0886260512471083>
- Golding, J. M. (1999). Intimate Partner Violence as a Risk Factor for Mental Disorders: A Meta-Analysis. *Journal of Family Violence*, 14, 34.
<http://www.ingentaconnect.com/content/klu/jofv/1999/00000014/00000002/00413367>
- Golinelli, D., Longshore, D., & Wenzel, S. L. (2009). Substance use and intimate partner violence: Clarifying the relevance of women's use and partners' use. *Journal of Behavioral Services & Research*, 36, 199–211. DOI:10.1007/s11414-008-9114-6.
- Goodman, L., Dutton, M. A., Vankos, N., & Weinfurt, K. (2005). Women's resources and use of strategies as risk and protective factors for reabuse over time. *Violence Against Women*, 11, 311–336. DOI:10.1177/1077801204273297
- Graham-Bermann, S. A. (2011). *The Moms' Empowerment Program: A Training Manual*. Department of Psychology, University of Michigan.
- Graham-Bermann, S. A., & Miller, L. E. (2013). Intervention to reduce traumatic stress following intimate partner violence: An efficacy trial. *Psychodynamic Psychiatry*, 41, 329–349.
- Graham-Bermann, S., Sularz, A. R., & Howell, K. H. (2011). Additional adverse events among women exposed to intimate partner violence: Frequency and impact. *Psychology of Violence*, 1, 136–149. DOI:10.1037/a0022975
- Graham, J. W., Olchowski, A. E., & Gilreath, T. D. (2007). How many imputations are really needed? Some practical clarifications of multiple imputation theory. *Prevention Science*, 8,

206–213. DOI:10.1007/s11121-007-0070-9

Grogan-Kaylor, A., Galano, M. M., Howell, K. H., Miller-Graff, L., & Graham-Bermann, S. A.

(2016). Reductions in parental use of corporal punishment on pre-school children following participation in the moms' empowerment program. *Journal of Interpersonal Violence*, 1–20. DOI:10.1177/0886260516651627

Gros, D. F., Stauffacher Gros, K., & Simms, L. J. (2010). Relations between anxiety symptoms and relational aggression and victimization in emerging adults. *Cognitive Therapy and Research*, 34, 134–143. DOI:10.1007/s10608-009-9236-z

Gu, J., Strauss, C., Bond, R., & Cavanagh, K. (2015). How do mindfulness-based cognitive therapy and mindfulness-based stress reduction improve mental health and wellbeing? A systematic review and meta-analysis of mediation studies. *Clinical Psychology Review*, 37, 1–12. DOI:10.1016/j.cpr.2015.01.006

Halpern, C. T., Spriggs, A. L., Martin, S. L., & Kupper, L. L. (2009). Patterns of Intimate Partner Violence Victimization from Adolescence to Young Adulthood in a Nationally Representative Sample. *Journal of Adolescent Health*, 45, 508–516. DOI:10.1016/j.jadohealth.2009.03.011

Hamby, S. (2016). Self-Report measures that do not produce gender parity in intimate partner violence: A multi-study investigation. *Psychology of Violence*, 6, 323–335. DOI:10.1037/a0038207

Herman, J. L. (1992). Complex PTSD: A syndrome in survivors of prolonged and repeated trauma. *Journal of Traumatic Stress*, 5, 377–391. DOI:10.1007/BF00977235

Herrenkohl, T. I., Mason, W. A., Kosterman, R., Lengua, L. J., Hawkins, J. D., & Abbott, R. D. (2004). Pathways From Physical Childhood Abuse to Partner Violence in Young

- Adulthood. *Violence and Victims*, 19, 123–136. DOI:10.1891/vivi.19.2.123.64099
- Hirschel, D., & Hutchison, I. W. (2003). The voices of domestic violence victims: Predictors of victim preference for arrest and the relationship between preference for arrest and revictimization. *Crime and Delinquency*, 49, 313–336. DOI:10.1177/0011128702251067
- Hofeller, K. H. (1982). *Social, psychological, and situational factors in wife abuse*. R&E Research Associates.
- Hotaling, G. T., & Sugarman, D. B. (1986). An analysis of risk markers in husband to wife violence: The current state of knowledge. *Violence and Victims*, 1, 101–124.
<http://www.ncbi.nlm.nih.gov/pubmed/3154143>
- Howell, K. H., Miller, L. E., Lilly, M. M., Burlaka, V., Grogan-Kaylor, A. C., & Graham-Bermann, S. A. (2015). Strengthening positive parenting through intervention: Evaluating the Moms' Empowerment Program for women experiencing intimate partner violence. *Journal of Interpersonal Violence*, 30, 232–252. DOI:10.1177/0886260514533155
- Illangasekare, S., Burke, J., Chander, G., & Gielen, A. (2013). The syndemic effects of intimate partner violence, HIV/AIDS, and substance abuse on depression among low-income Urban women. *Journal of Urban Health*, 90, 934–947. DOI:10.1007/s11524-013-9797-8
- Iverson, K. M., Gradus, J. L., Resick, P. a, Suvak, M. K., Smith, K. F., & Monson, C. M. (2011). Cognitive-Behavioral Therapy for PTSD and depression symptoms reduces risk for future intimate partner violence among interpersonal trauma survivors. *Journal of Consulting and Clinical Psychology*, 79, 193–202. DOI:10.1037/a0022512
- Iverson, K. M., Jimenez, S., Harrington, K. M., & Resick, P. A. (2011). The contributions of childhood family violence on later intimate partner violence among robbery victims. *Violence and Victims*, 26, 73–88. DOI:10.1891/0886-6708.26.1.73

- Iverson, K. M., Litwack, S. D., Pineles, S. L., Suvak, M. K., Vaughn, R. A., & Resick, P. A. (2013). Predictors of Intimate Partner Violence Revictimization: The Relative Impact of Distinct PTSD Symptoms, Dissociation, and Coping Strategies. *Journal of Traumatic Stress, 26*, 102–110. <http://doi.wiley.com/10.1002/jts.21781>
- Iverson, K., McLaughlin, K., Adair, K., & Monson, C. (2014). Anger-Related Dysregulation as a factor linking childhood physical abuse and interparental violence to intimate partner violence experiences. *Violence and Victims, 29*, 564–578.
DOI:10.1016/j.biotechadv.2011.08.021.Secreted
- Jackson, K. M., Sher, K. J., & Schulenberg, J. E. (2008). Conjoint Developmental Trajectories of Young Adult Substance Use. *Alcoholism: Clinical & Experimental Research, 32*, 723–737.
DOI:10.1111/j.1530-0277.2008.00643.x.Conjoint
- Jaffe, A. E., Cranston, C. C., & Shadlow, J. O. (2012). Parenting in females exposed to intimate partner violence and childhood sexual abuse. *Journal of Child Sexual Abuse, 21*, 684–700.
DOI:10.1080/10538712.2012.726698
- James, L., Brody, D., & Hamilton, Z. (2013). Risk Factors for Domestic Violence During Pregnancy: A Meta-Analytic Review. *Violence and Victims, 28*, 359–380.
DOI:10.1891/0886-6708.vv-d-12-00034
- Jankowski, M. K., Leitenberg, H., Henning, K., & Coffey, P. (2002). Parental caring as a possible buffer against sexual revictimization in young adult survivors of child sexual abuse. *Journal of Traumatic Stress, 15*, 235–244. DOI:10.1023/A:1015259412746
- Jewkes, R. (2002). Intimate partner violence: Causes and prevention. *Lancet, 359*, 1423–1429.
- Johnson, M. P. (1995). Patriarchal Terrorism and Common Couple Violence: Two Forms of Violence against Women. *Journal of Marriage and the Family, 57*, 283.

DOI:10.2307/353683

Johnson, M. P. (2008). *A typology of domestic violence: Intimate terrorism, violent resistance, and situational couple violence*. Northeastern University Press.

Johnston, L., O'Malley, P., Bachman, J., & Schulenberg, J. (2013). *Monitoring the Future national survey results on drug use, 1975-2012: Volume 2, College students and adult ages 19-50*.

Jouriles, E. N., Choi, H. J., Rancher, C., & Temple, J. R. (2017). Teen Dating Violence Victimization, Trauma Symptoms, and Revictimization in Early Adulthood. *Journal of Adolescent Health, 61*, 115–119. DOI:10.1016/j.jadohealth.2017.01.020

Kabat-Zinn, J. (2003). Mindfulness-based interventions in context: Past, present, and future. *Clinical Psychology: Science and Practice, 10*, 144–156. DOI:10.1093/clipsy/bpg016

Kantor, G K, & Asdigian, N. (1997). When women are under the influence. Does drinking or drug use by women provoke beatings by men. In *Recent developments in alcoholism, Volume 13: alcohol and violence* (Vol. 13, pp. 315–336). DOI:10.1007/0-306-47141-8

Kantor, G K, Jasinski, J. L., & Aldarondo, E. (1994). Sociocultural status and incidence of marital violence in Hispanic families. *Violence and Victims, 9*, 207–222.
<https://www.ncbi.nlm.nih.gov/pubmed/7647043>

Kantor, Glenda Kaufman, & Straus, M. A. (1987). The “Drunken Bum” Theory of Wife Beating. *Social Problems, 34*, 213–230. DOI:10.1525/sp.1987.34.3.03a00010

Karaca-Mandic, P., Norton, E. C., & Dowd, B. (2012). Interaction terms in nonlinear models. *Health Services Research, 47*, 255–274. DOI:10.1111/j.1475-6773.2011.01314.x

Kaysen, D., Pantalone, D. W., Chawla, N., Lindgren, K. P., Clum, G. A., Lee, C., & Resick, P. (2008). Posttraumatic stress disorder, alcohol use, and physical health concerns. *Journal of*

- Behavioral Medicine*, 31, 115–125. DOI:10.1007/s10865-007-9140-5. Posttraumatic
- Kim, C. (2017). The impact of perceived childhood victimization and patriarchal gender ideology on intimate partner violence (IPV) victimization among Korean immigrant women in the USA. *Child Abuse & Neglect*, 70, 82–91. DOI:10.1016/j.chiabu.2017.05.010
- Kimerling, R., Alvarez, J., Pavao, J., Kaminski, A., & Baumrind, N. (2007). Epidemiology and Consequences of Women's Revictimization. *Women's Health Issues*, 17, 101–106. DOI:10.1016/j.whi.2006.12.002
- Klein, H. (2011). Using a syndemics theory approach to Studying HIV risk taking in a population of men who use the internet to find partners for unprotected sex. *American Journal of Men's Health*, 5, 466–476. DOI:10.1177/1557988311398472
- Krause, E. D., Kaltman, S., Goodman, L., & Dutton, M. A. (2006). Role of distinct PTSD symptoms in intimate partner reabuse: A prospective study. *Journal of Traumatic Stress*, 19, 507–516. DOI:10.1002/jts
- Kuhl, D. C., Warner, D. F., & Warner, T. D. (2015). Intimate Partner Violence Risk Among Victims of Youth Violence: Are Early Unions Bad, Beneficial, or Benign? *Criminology*, 53, 427–456. DOI:10.1111/1745-9125.12075
- Kuijpers, K. F., van der Knaap, L. M., & Lodewijks, I. a J. (2011). Victims' influence on intimate partner violence revictimization: a systematic review of prospective evidence. *Trauma, Violence & Abuse*, 12, 198–219. DOI:10.1177/1524838011416378
- Kuijpers, K. F., van der Knaap, L. M., & Winkel, F. W. (2012). PTSD symptoms as risk factors for intimate partner violence revictimization and the mediating role of victims' violent behavior. *Journal of Traumatic Stress*, 25, 179–186. DOI:10.1002/jts.21676
- Kuijpers, K. F., Van der Knaap, L. M., & Winkel, F. W. (2012a). Risk of Revictimization of

- Intimate Partner Violence: The Role of Attachment, Anger and Violent Behavior of the Victim. *Journal of Family Violence*, 27, 33–44. DOI:10.1007/s10896-011-9399-8
- Kuijpers, K. F., Van der Knaap, L. M., & Winkel, F. W. (2012b). Victims' influence on intimate partner violence revictimization: An empirical test of dynamic victim-related risk factors. *Journal of Interpersonal Violence*, 27, 1716–1742. DOI:10.1177/0886260511430389
- La Flair, L. N., Bradshaw, C. P., Storr, C. L., Green, K. M., Alvanzo, A. a H., & Crum, R. M. (2012). Intimate partner violence and patterns of alcohol abuse and dependence criteria among women: A latent class analysis. *Journal of Studies on Alcohol and Drugs*, 73, 351–360. DOI:10.15288/jsad.2012.73.351
- Lacey, K. K., McPherson, M. D., Samuel, P. S., Powell Sears, K., & Head, D. (2012). The impact of different types of intimate partner violence on the mental and physical health of women in different ethnic groups. *Journal of Interpersonal Violence*, 28, 359–385. DOI:10.1177/0886260512454743
- Lagdon, S., Armour, C., & Stringer, M. (2014). Adult experience of mental health outcomes as a result of intimate partner violence victimisation: A systematic review. *European Journal of Psychotraumatology*, 5. DOI:10.3402/ejpt.v5.24794
- Langhinrichsen-Rohling, J., Misra, T. A., Selwyn, C., & Rohling, M. L. (2012). Rates of Bidirectional Versus Unidirectional Intimate Partner Violence Across Samples, Sexual Orientations, and Race/Ethnicities: A Comprehensive Review. *Partner Abuse*, 3, 199–230. DOI:10.1891/1946-6560.3.2.199
- Lehrer, J. a, Buka, S., Gortmaker, S., & Shrier, L. a. (2006). Depressive symptomatology as a predictor of exposure to intimate partner violence among US female adolescents and young adults. *Archives of Pediatrics & Adolescent Medicine*, 160, 270–276.

DOI:10.1001/archpedi.160.3.270

Lehrner, A., & Allen, N. E. (2014). Construct validity of the conflict tactics scales: A mixed-method investigation of women's intimate partner violence. *Psychology of Violence, 4*, 477–490. DOI:10.1037/a0037404

Lewinsohn, P. M., Seeley, J. R., Roberts, R. E., & Allen, N. B. (1997). Center for Epidemiologic Studies Depression Scale (CES-D) as a screening instrument for depression among community-residing older adults. *Psychology and Aging, 12*, 277–287.

Liebschutz, J. M., & Rothman, E. F. (2012). Intimate-Partner Violence — What Physicians Can Do. *New England Journal of Medicine, 367*, 2071–2073. DOI:10.1056/nejmp1204278

Lipsey, M. W., Wilson, D. B., Cohen, M. A., & Derzon, J. H. (1997). Is there a causal relationship between alcohol use and violence? A synthesis of evidence. In M. Galanter (Ed.), *Recent developments in alcoholism, Volume 13: alcohol and violence* (pp. 245–282). Plenum Press.

Lipsky, S., Caetano, R., Field, C. A., & Larkin, G. L. (2005). Psychosocial and substance-use risk factors for intimate partner violence. *Drug and Alcohol Dependence, 78*, 39–47. DOI:10.1016/j.drugalcdep.2004.08.028

Lipsky, S., Caetano, R., Field, C. a, & Bazargan, S. (2005). The role of alcohol use and depression in intimate partner violence among black and Hispanic patients in an urban emergency department. *The American Journal of Drug and Alcohol Abuse, 31*, 225–242. DOI:10.1081/ADA-200047923

Lipsky, S., Caetano, R., & Roy-Byrne, P. (2009). Racial and ethnic disparities in police-reported intimate partner violence and risk of hospitalization among women. *Women's Health Issues, 19*, 109–118. DOI:10.1016/j.whi.2008.09.005

- Loxton, D., Dolja-Gore, X., Anderson, A. E., & Townsend, N. (2017). Intimate partner violence adversely impacts health over 16 years and across generations: A longitudinal cohort study. *PLoS ONE*, *12*, 1–10. DOI:10.1371/journal.pone.0178138
- Luke, D. (2004). *Multilevel modeling*. Sage Publications.
- Martin, S.L., Tsui, A. O., Maitra, K., & Marinshaw, R. (1999). Domestic Violence in Northern India. *American Journal of Epidemiology*, *150*.
- Martin, Sandra L, Moracco, K. E., Chang, J. C., Council, C. L., & Dulli, L. S. (2008). Substance abuse issues among women in domestic violence programs: findings from North Carolina. *Violence against Women*, *14*, 985–997. DOI:10.1177/1077801208322103
- Martino, S. C., Collins, R. L., & Ellickson, P. L. (2005). Cross-lagged relationships between substance use and intimate partner violence among a sample of young adult women. *Journal of Studies on Alcohol*, *66*, 139–148. DOI:10.15288/jsa.2005.66.139
- Messing, J. T., Flair, L. La, Cavanaugh, C. E., Kanga, M. R., & Campbell, J. C. (2012). Testing Posttraumatic Stress as a mediator of childhood trauma and adult intimate partner violence victimization. *Journal of Aggression, Maltreatment & Trauma*, *21*, 792–811. DOI:10.1080/10926771.2012.686963
- Meyer, J. P., Springer, S. A., & Altice, F. L. (2011). Substance abuse, violence, and HIV in women: A literature review of the syndemic. *Journal of Women's Health*, *20*, 991–1006. DOI:10.1089/jwh.2010.2328
- Miller-Graff, L., & Graham-Bermann, S. (2016). Individual- and Community-level Predictors of Victimization Frequency in a Sample of Women Exposed to IPV. *Psychology of Violence*, *6*, 172–181. DOI:10.1037/a0038176
- Miller, L. E., Howell, K. H., & Graham-Bermann, S. A. (2014). The Effect of an Evidence-

- Based Intervention on Women's Exposure to Intimate Partner Violence. *American Journal of Orthopsychiatry*, 84, 321–328.
- Miller, W. C., Anton, H. A., & Townson, A. F. (2008). Measurement properties of the CESD scale among individuals with spinal cord injury. *Spinal Cord*, 46, 287–292.
DOI:10.1038/sj.sc.3102127
- Moffitt, T. E., Robins, R. W., & Caspi, A. (2001). A Couples Analysis of Partner Abuse with Implications for Abuse-Prevention Policy. *Criminology and Public Policy*, 1, 5–36.
- Murphy, L. M. (2011). Childhood and adolescent violent victimization and the risk of young adult intimate partner violence victimization. *Violence and Victims*, 26, 593–607.
DOI:10.1891/0886-6708.26.5.593
- National Center for Injury Prevention and Control. (2003). *Costs of intimate partner violence against women in the United States*.
<http://scholar.google.com/scholar?hl=en&btnG=Search&q=intitle:Costs+of+Intimate+Partner+Violence+Against+Women+in+the+United+States#1>
- Ngo, Q. M., Ramirez, J. I., Stein, S. F., Cunningham, R. M., Chermack, S. T., Singh, V., & Walton, M. A. (2018). Understanding the Role of Alcohol, Anxiety, and Trait Mindfulness in the Perpetration of Physical and Sexual Dating Violence in Emerging Adults. *Violence Against Women*, 24, 1166–1186. DOI:10.1177/1077801218781886
- Nowotny, K. M., & Graves, J. L. (2013). Substance use and intimate partner violence victimization among White, African American, and Latina women. *Journal of Interpersonal Violence*, 28, 3301–3318. <http://www.ncbi.nlm.nih.gov/pubmed/23946141>
- Ogden, P., & Fisher, J. (2015). *Sensorimotor psychotherapy: Interventions for trauma and attachment (Norton series on interpersonal neurobiology)*. WW Norton & Company.

- Ørke, E. C., Vatnar, S. K. B., & Bjørkly, S. (2018). Risk for Revictimization of Intimate Partner Violence by Multiple Partners: a Systematic Review. *Journal of Family Violence*. DOI:10.1007/s10896-018-9952-9
- Ouellet-Morin, I., Fisher, H. L., York-Smith, M., Fincham-Campbell, S., Moffitt, T. E., & Arseneault, L. (2015). Intimate partner violence and new-onset depression: A longitudinal study of women's childhood and adult histories of abuse. *Depression and Anxiety*, 32, 316–324. <http://doi.wiley.com/10.1002/da.22347>
- Palazzolo, K. E., Roberto, A. J., & Babin, E. A. (2010). The relationship between parents' verbal aggression and young adult children's intimate partner violence victimization and perpetration. *Health Communications*, 25, 357–364. DOI:10.1080/10410231003775180
- Palmetto, N., Davidson, L. L., Breitbart, V., & Rickert, V. I. (2013). Predictors of Physical Intimate Partner Violence in the Lives of Young Women: Victimization, Perpetration, and Bidirectional Violence. *Violence and Victims*, 28, 103–121. DOI:10.1891/0886-6708.28.1.103
- Parker, E. M., Debnam, K., Pas, E. T., & Bradshaw, C. P. (2016). Exploring the Link Between Alcohol and Marijuana Use and Teen Dating Violence Victimization Among High School Students: The Influence of School Context. *Health Education and Behavior*, 43, 528–536. DOI:10.1177/1090198115605308
- Perez, S., & Johnson, D. M. (2008). PTSD compromises battered women's future safety. *Journal of Interpersonal Violence*, 23, 635–651. DOI:10.1177/0886260507313528
- Peterson, C., Kearns, M. C., McIntosh, W. L. K. W., Estefan, L. F., Nicolaidis, C., McCollister, K. E., Gordon, A., & Florence, C. (2018). Lifetime Economic Burden of Intimate Partner Violence Among U.S. Adults. *American Journal of Preventive Medicine*, 55, 433–444.

DOI:10.1016/j.amepre.2018.04.049

Poole, N., Greaves, L., Jategaonkar, N., McCullough, L., & Chabot, C. (2008). Substance use by women using domestic violence shelters. *Substance Use & Misuse*, 43, 1129–1150.

DOI:10.1080/10826080801914360

Powers, M. B., Gillihan, S. J., Rosenfield, D., Jerud, A. B., & Foa, E. B. (2012). Reliability and validity of the PDS and PSS-I among participants with PTSD and alcohol dependence.

Journal of Anxiety Disorders, 26, 617–623. DOI:10.1016/j.janxdis.2012.02.013

Public Health Action Support Team. (2019). *Public Health Textbook*.

Radloff, L. S. (1977). The CES-D Scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement*, 1, 385–401.

DOI:10.1177/014662167700100306

Radloff, L. S., & Teri, L. (1986). Use of the Center for Epidemiological Studies Depression Scale with older adults. *Clinical Gerontologists*, 5, 119–136.

Raudenbush, S. W., & Bryk, A. S. (2002). *Hierarchical linear models: Applications and data analysis methods*. Sage Publications.

Recklitis, C. J., Parsons, S. K., Shih, M.-C., Mertens, A., Robison, L. L., & Zeltzer, L. (2006). Factor structure of the Brief Symptom Inventory--18 in adult survivors of childhood cancer:

Results from the Childhood Cancer Survivor Study. *Psychological Assessment*, 18, 22–32.

DOI:10.1037/1040-3590.18.1.22

Reichel, D. (2017). Determinants of intimate partner violence in Europe: The role of socioeconomic status, Inequality, and partner behavior. *Journal of Interpersonal Violence*,

32, 1853–1873. DOI:10.1177/0886260517698951

Reingle, J. M., Staras, S. A. S., Jennings, W. G., Branchini, J., & Maldonado-Molina, M. M.

- (2012). The Relationship Between Marijuana Use and Intimate Partner Violence in a Nationally Representative, Longitudinal Sample. *Journal of Interpersonal Violence*, 27, 1562–1578. DOI:10.1177/0886260511425787
- Renner, L. M., & Whitney, S. D. (2012). Risk factors for unidirectional and bidirectional intimate partner violence among young adults. *Child Abuse and Neglect*, 36, 40–52. DOI:10.1016/j.chiabu.2011.07.007
- Rennison, C., & Rand, M. R. (2003). Nonlethal intimate partner violence against women: a comparison of three age cohorts. *Violence Against Women*, 9, 1417–1428. DOI:10.1177/1077801203259232
- Rodriguez-Menés, J., Puig, D., & Sobrino, C. (2014). Poly- and Distinct- Victimization in Histories of Violence Against Women. *Journal of Family Violence*, 29, 849–858. DOI:10.1007/s10896-014-9638-x
- Roy, M. (1982). *The abusive partner: An analysis of domestic battering*. Van Nostrand Reinhold.
- Royston, P. (2004). Multiple imputation of missing values. *The Stata Journal*, 4, 227–241. DOI:10.1111/j.1541-0420.2010.01538.x
- Schafer, J. L. (1999). Multiple imputation: A primer. *Statistical Methods in Medical Research*, 8, 3–15.
- Schauer, M., & Elbert, T. (2010). Dissociation following traumatic stress etiology and treatment. *Journal of Psychology*, 218, 109–127. DOI:10.1027/0044-3409/a000018
- Schore, A. (2001). Effects of a secure attachment relationship on right brain development, affect regulation, and infant mental health. *Infant Mental Health Journal*, 22, 201–269. DOI:10.1002/1097-0355(200101/04)22:1<7::AID-IMHJ2>3.0.CO;2-N
- Schore, A. N. (2002). Dysregulation of the right brain: A fundamental mechanism of traumatic

- attachment and the psychopathogenesis of posttraumatic stress disorder. *Australian and New Zealand Journal of Psychiatry*, 36, 9–30.
- Schumacher, J. A., & Holt, D. J. (2012). Domestic violence shelter residents' substance abuse treatment needs and options. *Aggression and Violent Behavior*, 17, 188–197.
DOI:10.1016/j.avb.2012.01.002
- Schwartz, R. C. (2013). Moving from acceptance toward transformation with internal family systems therapy (IFS). *Journal of Clinical Psychology*, 69, 805–816.
DOI:10.1002/jclp.22016
- Senn, T. E., Carey, M. P., & Vanable, P. A. (2010). The intersection of violence, substance use, depression, and STDs: Testing of a syndemic pattern among patients attending an urban STD clinic. *Journal of the National Medical Association*, 102, 614–620.
DOI:10.1016/S0027-9684(15)30639-8
- Shapiro, S. L., Brown, K. W., Thoresen, C., & Plante, T. G. (2011). The moderation of Mindfulness-based stress reduction effects by trait mindfulness: Results from a randomized controlled trial. *Journal of Clinical Psychology*, 67, 267–277. DOI:10.1002/jclp.20761
- Sheeran, T., & Zimmerman, M. (2002). Screening for Posttraumatic Stress Disorder in a general psychiatric outpatient setting. *Journal of Counseling and Clinical Psychology*, 70, 961–966.
DOI:10.1037//0022-006X.70.4.000
- Shorey, R. C., Moore, T. M., McNulty, J. K., & Stuart, G. L. (2016). Do alcohol and marijuana increase the risk for female dating violence victimization? A prospective daily diary investigation. *Psychology of Violence*, 6, 509. DOI:doi:10.1037/a0039943
- Shorey, R. C., Stuart, G. L., & Cornelius, T. L. (2011). Dating Violence and Substance Use in College Students: A Review of the Literature. *Aggression and Violent Behavior*, 16, 541–

550. DOI:10.1016/j.avb.2011.08.003.Dating

Shorey, Ryan C., Brasfield, H., Febres, J., & Stuart, G. L. (2011). An examination of the association between difficulties with emotion regulation and dating violence perpetration. *Journal of Aggression, Maltreatment and Trauma*, 20, 870–885.

DOI:10.1080/10926771.2011.629342

Shorey, Ryan C., Cornelius, T. L., & Bell, K. M. (2008). A critical review of theoretical frameworks for dating violence: Comparing the dating and marital fields. *Aggression and Violent Behavior*, 13, 185–194. DOI:10.1016/j.avb.2008.03.003

Shorey, Ryan C., Rhatigan, D. L., Fite, P. J., & Stuart, G. L. (2011). Dating violence victimization and alcohol problems: an examination of the stress-buffering hypothesis for perceived support. *Partner Abuse*, 2, 31–45. DOI:10.1891/1946

Simmons, S. B., Knight, K. E., & Menard, S. (2018). Long-Term Consequences of Intimate Partner Abuse on Physical Health, Emotional Well-Being, and Problem Behaviors. In *Journal of Interpersonal Violence* (Vol. 33, Issue 4). DOI:10.1177/0886260515609582

Singer, J. D., & Willett, J. B. (2003). *Applied longitudinal data analysis: Modeling change and event occurrence*. Oxford University Press.

Singer, M. (2009). *Introduction to syndemics: A critical systems approach to public and community health*. John Wiley & Sons.

Singer, Merrill, Bulled, N., & Ostrach, B. (2020). Whither syndemics?: Trends in syndemics research, a review 2015–2019. *Global Public Health*, 15, 943–955.

DOI:10.1080/17441692.2020.1724317

Singer, Merrill, Bulled, N., Ostrach, B., & Mendenhall, E. (2017). Syndemics and the biosocial conception of health. *The Lancet*, 389, 941–950. DOI:10.1016/S0140-6736(17)30003-X

- Singer, Merrill, & Clair, S. (2003). Syndemics and public health: Reconceptualizing disease in bio-social context. *Medical Anthropology Quarterly*, 17, 423–441.
<http://www.ncbi.nlm.nih.gov/pubmed/14716917>
- Smith, L. S., & Stover, C. S. (2016). The Moderating Role of Attachment on the Relationship Between History of Trauma and Intimate Partner Violence Victimization. *Violence Against Women*, 22, 745–764. DOI:10.1177/1077801215610863
- Smith, S. G., Zhang, X., Basile, K. C., Merrick, M. T., Wang, J., Kresnow, M. J., & Chen, J. (2018). *The national intimate partner and sexual violence survey: 2015 data brief*.
- Sonis, J. (2008). Posttraumatic Stress Disorder does not Increase Recurrent Intimate Partner Violence. *Journal of Psychological Trauma*, 6, 27–48. DOI:10.1080/19322880802096459
- Sonis, J., & Langer, M. (2008). Risk and protective factors for recurrent intimate partner violence in a cohort of low-income inner-city women. *Journal of Family Violence*, 23, 529–538. DOI:10.1007/s10896-008-9158-7
- Spencer, C. M., Stith, S. M., & Cafferky, B. (2019). Risk markers for physical intimate partner violence victimization: A meta-analysis. *Aggression and Violent Behavior*, 44, 8–17.
DOI:10.1016/j.avb.2018.10.009
- Spencer, C., Mallory, A. B., Cafferky, B. M., Kimmes, J. G., Beck, A. R., & Stith, S. M. (2019). Mental health factors and intimate partner violence perpetration and victimization: A meta-analysis. *Psychology of Violence*, 9, 1–17. DOI:10.1037/vio0000156
- StataCorp. (2021a). *Stata 17 base reference manual*. Stata Press.
- StataCorp. (2021b). *Stata Statistical Software: Release 17*. StataCorp LLC.
- Steele, C. M., & Josephs, R. A. (1988). Drinking Your Troubles Away II: An Attention-Allocation Model of Alcohol's Effect on Psychological Stress. *Journal of Abnormal*

Psychology, 97, 196–205. DOI:10.1037/0021-843X.97.2.196

Stein, S. F., Grogan-Kaylor, A. A., Galano, M. M., Clark, H. M., & Graham-Bermann, S. A. (2019). The Social and Individual Characteristics of Women Associated With Engagement With Multiple Intimate Violent Partners. *Journal of Interpersonal Violence*, 34, 4572–4596. DOI:10.1177/0886260516676477

Stein, S.F., Galano, M. M., Grogan-Kaylor, A. C., Clark, H. M., Ribaud, J. M., & Graham-Bermann, S. A. (n.d.). *Predictors of intimate partner violence victimization by multiple partners over eight years*.

Stein, Sara F., Grogan-Kaylor, A. C., Galano, M. M., Clark, H. M., & Graham-Bermann, S. A. (2018). Contributions to Depressed Affect in Latina Women: Examining the Effectiveness of the Moms' Empowerment Program. *Journal of Interpersonal Violence*. DOI:10.1177/0886260518760005

Stith, S. M., Smith, D. B., Penn, C. E., Ward, D. B., & Tritt, D. (2004). Intimate partner physical abuse perpetration and victimization risk factors: A meta-analytic review. In *Aggression and Violent Behavior* (Vol. 10, Issue 1). DOI:10.1016/j.avb.2003.09.001

Straus, M.A. (2012). Blaming the messenger for the bad news about partner violence by women: The methodological, theoretical, and value basis of purported invalidity of the Conflict Tactics Scales. *Behavioral Sciences and the Law*, 30, 538–556. DOI:10.1002/bsl.2023

Straus, Murray A. (1979). Measuring intrafamily conflict and violence: The Conflict Tactics Scales. *Journal of Marriage and the Family*, 10, 75–88.

Straus, Murray A., Hamby, S. L., Boney-McCoy, S., & Sugarman, D. B. (1996). The Revised Conflict Tactics Scales (CTS2): Development and preliminary psychometric data. *Journal of Family Issues*, 17, 283–316. DOI:0803973233

- Straus, Murray A. (1973). A general systems theory approach to a theory of violence between family members. *Social Science Information*, 12, 105–125.
- Stuart, G. L., Moore, T. M., Elkins, S. R., Timothy, J., Farrell, O., Temple, J. R., Ramsey, S., & Shorey, R. C. (2013). The temporal association between substance use and intimate partner violence among women arrested for domestic violence. *Journal of Consulting and Clinical Psychology*, 81, 1–18. DOI:10.1037/a0032876
- Stuart, G., Moore, T., Ramsey, S., & Kahler, C. W. (2004). Hazardous drinking and relationship violence perpetration and victimization in women arrested for domestic violence. *Journal of Studies on Alcohol*, 65, 46–53. DOI:10.15288/jsa.2004.65.46
- Substance Abuse and Mental Health Service Administration. (2014). *Results from the 2013 National survey on drug use and health: summary of findings. NSDUH Series H-48, HHS Publication no. (SMA)*.
- Suglia, S. F., Duarte, C. S., & Sandel, M. T. (2011). Housing quality, housing instability, and maternal mental health. *Journal of Urban Health*, 88, 1105–1116. DOI:10.1007/s11524-011-9587-0
- Sullivan, K. A., Messer, L. C., & Quinlivan, E. B. (2015). Substance abuse, violence, and HIV/AIDS (SAVA) syndemic effects on viral suppression among HIV positive women of color. *AIDS Patient Care and STDs*, 29 Suppl 1, S42-8. DOI:10.1089/apc.2014.0278
- Tanskanen, M., & Kivivuori, J. (2021). Understanding intimate partner violence in context: social and community correlates of special and general victimization. *Nordic Journal of Criminology*, 22, 72–89. DOI:10.1080/2578983x.2021.1904605
- Taylor, C. A., Boris, N. W., Clum, G. A., Rice, J. C., & Zeanah, C. H. (2008). Cumulative experiences of violence among high-risk urban youth. *Journal of Interpersonal Violence*,

23, 1618–1635. DOI:10.1177/0886260508314323

Temple, J. R., Weston, R., Stuart, G. L., & Marshall, L. L. (2008). The longitudinal association between alcohol use and intimate partner violence among ethnically diverse community women. *Addictive Behaviors*, 33, 1244–1248. DOI:10.1016/j.addbeh.2008.05.005

Testa, M. (2004). The Role of Substance Use in Male-to-Female Physical and Sexual Violence. *Journal of Interpersonal Violence*, 19, 1494–1505. DOI:10.1177/0886260504269701

Testa, M., Livingston, J. A., & Leonard, K. E. (2004). Women's substance use and experiences of intimate partner violence: A longitudinal investigation among a community sample. *Addictive Behaviors*, 28, 1649–1664. DOI:10.1016/j.addbeh.2003.08.040

Trevillion, K., Oram, S., Feder, G., & Howard, L. M. (2012). Experiences of Domestic Violence and Mental Disorders: A Systematic Review and Meta-Analysis. *PLoS ONE*, 7. DOI:10.1371/journal.pone.0051740

Trickett, P. K., Noll, J. G., & Putnam, F. W. (2011). The impact of sexual abuse on female development: Lessons from a multigenerational, longitudinal research study. *Developmental Psychology*, 23, 453–476. DOI:10.1007/s11103-011-9767-z.Placid

Tsai, A. C., Mendenhall, E., Trostle, J. A., & Kawachi, I. (2017). Co-occurring epidemics, syndemics, and population health. *Lancet*, 389, 978–982. DOI:10.1016/S0140-6736(17)30403-8.Co-occurring

Van der Kolk, B. A. (1994). The body keeps the score: Memory and the evolving psychobiology of posttraumatic stress. *Harvard Review of Psychiatry*, 1, 253–265. DOI:10.3109/10673229409017088

Van der Kolk, B. A. (2015). *The body keeps the score: Brain, mind, and body in the healing of trauma*. Penguin Books.

- Vatnar, S. K. B., & Bjørkly, S. (2008). An interactional perspective of intimate partner violence: An in-depth semi-structured interview of a representative sample of help-seeking women. *Journal of Family Violence, 23*, 265–279. DOI:10.1007/s10896-007-9150-7
- Vladutiu, C. J., Martin, S. L., & Macy, R. J. (2011). College- or university-based sexual assault prevention programs: A review of program outcomes, characteristics, and recommendations. *Trauma, Violence, and Abuse, 12*, 67–86.
DOI:10.1177/1524838010390708
- Walton, M. A., Murray, R., Cunningham, R. M., Chermack, S. T., Barry, K. L., Booth, B. M., Ilgen, M. A., Wojnar, M., & Blow, F. C. (2009). Correlates intimate partner violence among men and women in an inner city emergency department. *Journal of Addictive Disease, 28*, 83–88. DOI:10.1016/j.jdiacomp.2008.01.002.
- Whitaker, D. J., Haileyesus, T., Swahn, M., & Saltzman, L. S. (2007). Differences in frequency of violence and reported injury between relationships with reciprocal and nonreciprocal intimate partner violence. *American Journal of Public Health, 97*, 941–947.
DOI:10.2105/AJPH.2005.079020
- White, H. R., & Chen, P.-H. (2002). Problem drinking and intimate partner violence. *Journal of Studies on Alcohol, 63*, 205–214. DOI:10.15288/jsa.2002.63.205
- White, H. R., Jackson, K. M., & Loeber, R. (2009). Developmental Sequences and Comorbidity of Substance Use and Violence. In M. D. Krohn, A. J. Lizotte, & G. P. Hall (Eds.), *Handbook on Crime and Deviance* (pp. 209–224). DOI:10.1007/978-1-4419-0245-0
- Whitfield, C. L., Anda, R. F., Dube, S. R., & Felitti, V. J. (2003). Violent Childhood Experiences and the Risk of Intimate Partner Violence in Adults: Assessment in a Large Health Maintenance Organization. *Journal of Interpersonal Violence, 18*, 166–185.

DOI:10.1177/0886260502238733

- Widom, C. S., Czaja, S., & Dutton, M. A. (2014). Child abuse and neglect and intimate partner violence victimization and perpetration: A prospective investigation. *Child Abuse & Neglect*, 38, 650–663. DOI:10.1016/j.chiabu.2013.11.004.Child
- Williams, D. R., Priest, N., & Anderson, N. B. (2016). Understanding associations among race, socioeconomic status, and health: Patterns and prospects. *Health Psychology*, 35, 407. DOI:10.1002/cncr.27633.Percutaneous
- Yurasek, A. M., Aston, E. R., & Metrik, J. (2017). Co-use of Alcohol and Cannabis: A Review. *Current Addiction Reports*, 4, 184–193. DOI:10.1007/s40429-017-0149-8

Appendices

Appendix A: Demographic Questionnaire (Study 1 & 2)

Date of last interview:

Previously completed interviews (circle all that apply): T1 – T2 – T3

Date of this interview:

Child's Birthdate:

Child's Sex:

Child's Age:

Mom's Age:

1) We want to get a sense of who are the people in your child's life. Please tell us who are the people (family and friends, including parents, siblings, partners/boyfriends, other relatives, etc.) whom your child sees on a regular basis. For each, tell if you think your child would identify that person as a significant person in their life. Please indicate if that person is a member of the household (living in your home).

	Relationship to child (e.g., mom, dad, family, friend)	Sex	Age	Lives in home with child?	
1	Mother			Yes	No
2				Yes	No
3				Yes	No
4				Yes	No
5				Yes	No
6				Yes	No
7				Yes	No
8				Yes	No
9				Yes	No
10				Yes	No

2) Your current relationship status (check one):

- | | |
|---|---|
| <input type="checkbox"/> Single
<input type="checkbox"/> Living with partner
<input type="checkbox"/> Married
<input type="checkbox"/> Separated (How long? _____) | <input type="checkbox"/> Widowed
<input type="checkbox"/> Divorced
<input type="checkbox"/> Remarried |
|---|---|

3) What category best describes you and your child's race or ethnicity?

- | you | child | |
|--------------------------|--------------------------|---------------------------------|
| <input type="checkbox"/> | <input type="checkbox"/> | Native American |
| <input type="checkbox"/> | <input type="checkbox"/> | Asian |
| <input type="checkbox"/> | <input type="checkbox"/> | Black, African-American |
| <input type="checkbox"/> | <input type="checkbox"/> | Latino, Hispanic-American |
| <input type="checkbox"/> | <input type="checkbox"/> | Biracial (mixed): specify _____ |
| <input type="checkbox"/> | <input type="checkbox"/> | White |
| <input type="checkbox"/> | <input type="checkbox"/> | Other _____ |

4) What is the highest level of education that you have completed?

- | | |
|--|---|
| <input type="checkbox"/> Grade school or less | <input type="checkbox"/> College degree |
| <input type="checkbox"/> Some high school | <input type="checkbox"/> Some graduate school |
| <input type="checkbox"/> High school degree/GED | <input type="checkbox"/> Graduate degree |
| <input type="checkbox"/> Some college or vocational school | |

5) Are you working at this time?

- | | |
|------------------------------|-----------------------|
| <input type="checkbox"/> Yes | Hours per week? _____ |
| <input type="checkbox"/> No | |

6) What job do you do (i.e., what is your job title)? _____

7) What was your total household income last month? \$ _____

8) How many times have you moved since the last interview? _____

9) Have you ever gone to a “safe house” or women’s shelter for abused women since your last interview?

- | | |
|------------------------------|-----------------------------|
| <input type="checkbox"/> Yes | <input type="checkbox"/> No |
|------------------------------|-----------------------------|

10) If yes, how many times: _____

Appendix B: Revised Conflict Tactics Scale (Study 1 & 2)

No matter how well a couple gets along, there are times when they disagree, get annoyed with one another, want different things from each other, or just have spats or fights because they are in a bad mood, are tired, or are upset for some other reason. Couples also have many different ways of trying to settle their differences. This is a list of things that might happen when you have differences. Please tell us how many times these things have happened in the past year.

	1x	2x	3-5x	6-10x	11-20x	>20x	Never
1. My partner showed care for me even though we disagreed.	1	2	3	4	5	6	7
2. My partner explained his or her side of a disagreement to me.	1	2	3	4	5	6	7
3. My partner insulted or swore at me.	1	2	3	4	5	6	7
4. My partner threw something at me that could hurt.	1	2	3	4	5	6	7
5. My partner twisted my arm or hair.	1	2	3	4	5	6	7
6. You had a sprain, bruise or small cut because of a fight with your partner.	1	2	3	4	5	6	7
7. My partner showed respect for my feelings about an issue.	1	2	3	4	5	6	7
8. My partner made me have sex without a condom.	1	2	3	4	5	6	7
9. My partner pushed or shoved me.	1	2	3	4	5	6	7
10. My partner used force to make me have oral or anal sex.	1	2	3	4	5	6	7
11. My partner used a knife or gun on me.	1	2	3	4	5	6	7
12. You passed out from being hit on the head by your partner in a fight.	1	2	3	4	5	6	7
13. My partner called me fat or ugly.	1	2	3	4	5	6	7
14. My partner punched or hit me with something that could hurt.	1	2	3	4	5	6	7
15. My partner destroyed something that belonged to me.	1	2	3	4	5	6	7
16. You went to a doctor because of a fight with your partner.	1	2	3	4	5	6	7
17. My partner choked me.	1	2	3	4	5	6	7
18. My partner shouted or yelled at me.	1	2	3	4	5	6	7
19. My partner slammed me against a wall.	1	2	3	4	5	6	7
20. My partner was sure we could work it out.	1	2	3	4	5	6	7
21. You needed to see a doctor because of a fight with your partner, but didn't.	1	2	3	4	5	6	7
22. My partner beat me up.	1	2	3	4	5	6	7
23. My partner grabbed me.	1	2	3	4	5	6	7
24. My partner used force to make me have sex.	1	2	3	4	5	6	7
25. My partner stomped out of the room or house or yard during a disagreement.	1	2	3	4	5	6	7
26. My partner insisted that I have sex when I didn't want to (but did not use physical force).	1	2	3	4	5	6	7
27. My partner slapped me.	1	2	3	4	5	6	7
28. You had a broken bone from a fight with your partner.	1	2	3	4	5	6	7
29. My partner used threats to make me have oral or anal sex.	1	2	3	4	5	6	7
30. My partner suggested a compromise to a disagreement.	1	2	3	4	5	6	7
31. My partner burned or scalded me on purpose.	1	2	3	4	5	6	7
32. My partner insisted that I have oral or anal sex (but did not use physical force)	1	2	3	4	5	6	7
33. My partner accused me of being a lousy lover.	1	2	3	4	5	6	7
34. My partner did something to spite me.	1	2	3	4	5	6	7
35. My partner threatened to hit or throw something at me.	1	2	3	4	5	6	7
36. You still felt physical pain the next day because of a fight you had with your partner.	1	2	3	4	5	6	7
37. My partner kicked me.	1	2	3	4	5	6	7
38. My partner used threats to make me have sex.	1	2	3	4	5	6	7
39. My partner agreed to try a solution I suggested.	1	2	3	4	5	6	7
40. Are you currently living with a violent partner? Yes (1) No (0)							

- a. If yes, how long have you lived with this partner? _____
- b. If no, when was the last time that you lived with a violent partner, if ever? _____
- 41. How many violent partners have you had in your life? _____
- 42. How many violent partners have you had since the last time we interviewed you? _____
- 43. How old were you when you had your first violent partner? _____
- 44. How many non-violent intimate partners have you had in your lifetime? _____
- 44. How many non-violent intimate partners have you had since the last time we interviewed you? _____

Appendix C: Posttraumatic Diagnostic Scale (Study 1 & 2)

The next questions have to do with dealing with stressful situations that may have happened to you or you may have seen. It is OK to pass on any question you do not want to answer. Many people have lived through or witnessed a very stressful and traumatic event at some point in their lives. Indicate whether or not you or your child have experienced or witnessed each traumatic event since the last interview. We may ask you when was the last time that the event occurred.

1. Serious accident, fire, or explosion (for example, an industrial, farm, car, plane, or boating accident)
Me ____ Child ____ Both ____ No One ____ When _____
2. Natural disaster (for example, tornado, hurricane, flood, or major earthquake)
Me ____ Child ____ Both ____ No One ____ When _____
3. Non-sexual assault by a family member or someone you know (for example, being mugged, physically attacked, shot, stabbed, or held at gunpoint)
Me ____ Child ____ Both ____ No One ____ When _____
4. Non-sexual assault by a stranger (for example, being mugged, physically attacked, shot, stabbed, or held at gunpoint)
Me ____ Child ____ Both ____ No One ____ When _____
5. Sexual assault by a family member or someone you know (for example, rape or attempted rape)
Me ____ Child ____ Both ____ No One ____ When _____
6. Sexual assault by a stranger (for example, rape or attempted rape)
Me ____ Child ____ Both ____ No One ____ When _____
7. Military combat or a war zone
Me ____ Child ____ Both ____ No One ____ When _____
8. Sexual contact when you were younger than 18 with someone who was 5 or more years older than you (for example, contact with genitals, breasts)
Me ____ Child ____ Both ____ No One ____ When _____
8. Imprisonment (for example, prison inmate, prisoner of war, hostage)
Me ____ Child ____ Both ____ No One ____ When _____
10. Torture
Me ____ Child ____ Both ____ No One ____ When _____
11. Life-threatening illness
Me ____ Child ____ Both ____ No One ____ When _____
12. Other traumatic event (read examples below)
Me ____ Child ____ Both ____ No One ____ When _____

e.g., Attacked by an animal, Man-made disasters (crashes, fires, war), Witnessed another person being beaten, raped, threatened with serious harm, shot at, seriously wounded, or killed, Accidental burning, Near drowning, Hospitalization, emergency room visit, or invasive medical procedures, Kidnapped or Other event.

13. Explain if 'yes' to item 12: _____

Though you may have experienced a variety of traumatic events, we would like for you to respond to the following questions only in relation to physical and/or sexual assault that you've experienced from a partner.

14. Though you may have had many traumatic events occur with your partner, can you tell me which one you remember as the worst, or the one that has maybe stuck with you the most?

15. How long ago did that traumatic event happen? Or, when was the last time it happened? (mark ONE)

1. Less than 1 month
2. 1 to 3 months
3. 3 to 6 months
4. 6 months to 3 years
5. 3 to 5 years
6. More than 5 years

- | | | |
|---|-----------|----------|
| 16. During this traumatic event, were you physically injured? | _____ Yes | _____ No |
| 17. During this traumatic event, was someone else physically injured? | _____ Yes | _____ No |
| 18. Did you think that your life was in danger? | _____ Yes | _____ No |
| 19. Did you think that someone else's life was in danger? | _____ Yes | _____ No |
| 20. Did you feel helpless? | _____ Yes | _____ No |
| 21. Did you feel terrified? | _____ Yes | _____ No |

Below is a list of problems that people sometimes have after experiencing a traumatic event. Please choose an answer that best describes how often that problem has bothered you IN THE LAST MONTH:

0: not at all or only one time	2: 2-4 times a week/half the time
1: once a week or less/once in a while	3: 5 or more times a week/almost always

22. Having upsetting thoughts or images about the traumatic event that came into your head when you didn't want them to: _____
23. Having bad dreams or nightmares about the traumatic event: _____
24. Reliving the traumatic event, acting or feeling as if it was happening again: _____
25. Feeling emotionally upset when you were reminded of the traumatic event (for example, feeling scared, angry, sad, guilty, etc.): _____
26. Experiencing physical reactions when you were reminded of the traumatic event (for example, breaking out in a sweat, heart beating fast): _____
27. Trying not to think about, talk about, or have feelings about the traumatic event: _____
28. Trying to avoid activities, people, or places that remind you of the traumatic event: _____
29. Not being able to remember an important part of the traumatic event: _____
30. Having much less interest or participating much less often in important activities: _____
31. Feeling distant or cut off from people around you: _____

32. Feeling emotionally numb (for example, being unable to cry or unable to have loving feelings): _____
33. Feeling as if your future plans or hopes will not come true (for example, you will not have a career, marriage, children, or a long life): _____
34. Having trouble falling or staying asleep: _____
35. Feeling irritable or having fits of anger: _____
36. Having trouble concentrating (for example, drifting in and out of conversations, losing track of a story on television, forgetting what you read): _____
37. Being overly alert (for example, checking to see who is around you, being uncomfortable with your back to a door, etc.): _____
38. Being jumpy or easily startled (for example, when someone walks up behind you): _____
39. How long have you experienced the problems that you reported above? (may need to remind them of relevant symptoms) (Mark only ONE)
1. Less than 1 month 2. 1 to 3 months 3. More than 3 months
40. How long after the traumatic event did these problems begin? (Mark only ONE)
1. Less than 6 months 2. 6 or more months

Have the problems we just discussed interfered with any of the following areas of your life **DURING THE PAST MONTH**:

- | | |
|---|--------------------|
| 41. Work | _____ Yes _____ No |
| 42. Household chores and duties | _____ Yes _____ No |
| 43. Relationships with friends | _____ Yes _____ No |
| 44. Fun and leisure activities | _____ Yes _____ No |
| 45. Schoolwork | _____ Yes _____ No |
| 46. Relationships with your family | _____ Yes _____ No |
| 47. Sex life | _____ Yes _____ No |
| 48. General satisfaction with life | _____ Yes _____ No |
| 49. Overall level of function in all areas of your life | _____ Yes _____ No |

Appendix D: Center for Epidemiologic Studies Depression Scale (Study 1 & 2)

These questions are about how you, the parent, have been feeling within **the past week**. Please tell me how much of the time you have felt a certain way: 1= None of the time, 2= Some of the time, 3 = Occasionally or a moderate amount of the time, and 4 = Most or all of the time.

1= None of the time 2= Some of the time 3 = Occasionally 4 =Most or all of the time

- _____ 1. I was bothered by things that don't usually bother me.
- _____ 2. I did not feel like eating.
- _____ 3. I felt that I could not shake off the blues, even with help from family or friends.
- _____ 4. I felt that I was just as good as other people.
- _____ 5. I had trouble keeping my mind on what I was doing.
- _____ 6. I felt depressed.
- _____ 7. I felt that everything I did was an effort.
- _____ 8. I felt hopeful about the future.
- _____ 9. I thought my life had been a failure.
- _____ 10. I felt fearful.
- _____ 11. My sleep had been restless.
- _____ 12. I was happy.
- _____ 13. I talked less than usual.
- _____ 14. People were unfriendly.
- _____ 15. I felt lonely.
- _____ 16. I enjoyed life.
- _____ 17. I had crying spells.
- _____ 18. I felt sad.
- _____ 19. I felt that people disliked me.
- _____ 20. I could not "get going".
- _____ 21. Is the last week typical of how you have been feeling? **Yes or No**